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LECTURES

ON

F E V E R S .

BY

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UNIVERSITY OF THE CITY OF NEW YORK; CONSULTING PHYSICIAN TO THE CHARITY HOSPITAL
—TO THE BUREAU OF OUT-DOOR RELIEF—TO THE NORTH-WESTERN DISPENSARY—
TO THE CENTRAL DISPENSARY; LATE VISITING PHYSICIAN TO THE
BLACKWELL'S ISLAND FEVER HOSPITAL; VISITING PHYSICIAN TO
BELLEVUE HOSPITAL—TO THE MOUNT SINAI HOSPITAL,
ETC., ETC.

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PREFACE.

THESE lectures were delivered in the Medical Department of the University of the City of New York, to the Class of 1876-77.

With unimportant alterations, I now offer them as they were phonographically reported by Dr. Wm. M. Carpenter.

As in the preparation of my "Lectures on Diseases of the Lungs, Heart, and Kidneys," it has been my custom, after careful reading and close analysis of the subject of each lecture, to trust that the stimulus of the class would enable me to present the most recent views of acknowledged authorities, combined with the results of my own clinical observation and experience, in so simple, intelligible, and concise a manner that each student might master the prominent points.

I have adopted an etiological basis in the classification of fevers, and have endeavored to include in a few general classes all the numerous types described by different writers.

I have referred to theoretical questions only so far as was necessary in order to the proper understanding of subjects under consideration.

The Bibliography which accompanies these lectures includes those books, monographs, and theses which have been published since 1850, nearly all of which have been written, or are in circulation, in this country.

A few old books have been referred to, because they con-

tain many of the so-called new theories and modes of treating fevers.

My aim has been to give a summary of the literature of fevers in this country, and so much of foreign literature upon this subject as might be of interest and service to the student who desires to thoroughly investigate the subject of fevers. No notice has been taken of papers which have only appeared in medical journals.

These lectures are the result of careful study of the literature referred to in the Bibliography, combined with extensive clinical experience.

I have endeavored to be unbiassed in my statements of facts.

It is my purpose at some future time to publish, in similar form, lectures upon other infectious diseases.

42 WEST TWENTY-FIFTH STREET, August, 1877.

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MIASMATIC-CONTAGIOUS
FEVERS.

LECTURE I.

FEVERS.

Introduction.—Classification.—Typhoid Fever.—Morbid Anatomy.

GENTLEMEN :—We will commence this course of lectures with the study of those diseases which depend upon *morbid conditions of the blood*, produced by morbid agents developed exterior to the body of the affected. Such morbid agents may give rise, either directly or indirectly, to morbid processes : either by the changes which they produce in the blood, or by their action on the different organs and tissues of the body to which they are conveyed by the blood-vessels and lymphatics.

The class of morbid agents which will now especially engage our attention may be included under the general head of *viruses*.

By the term virus I mean a morbid substance which is developed either from animal or vegetable tissues in the process of decomposition, or from the excretions of diseased living beings. Many viruses are volatile, and may be conveyed either by air, by fluids, or by solids, and when so conveyed they become the means by which diseases known as contagious or infectious are transmitted. Some viruses are palpable poisons, and may be transmitted from the diseased to the healthy by inoculation.

When the virus which gives rise to a disease has its origin only in a living being, from whom it is excreted in an active

state, capable of conveyance from one person to another, then the disease which it produces is called contagious, and the virus is called a *contagion*.

If the morbid agent which has the power of developing disease has originated from decomposing organic matter, and has been diffused through the air or water, so that infection may have resulted without contact with one already diseased, the disease is called *miasmatic*, and the virus is called a *miasm*. For instance, intermittent fever is a miasmatic disease, while small-pox and measles are contagious diseases.

With our present knowledge of the nature and origin of viruses, we can make no classification, except that which is based on their differences of action. We speak of typhus, typhoid, and malarial poisons, but these different poisons have as yet no known physical or chemical properties by which we are able to distinguish one from another. We can only recognize their presence by the peculiar morbid phenomena which each has the power of developing in the animal economy.

The different diseases which are developed by the morbid processes excited by these different viruses are, at the present time, classed under the head of *infectious diseases*, and the influence of these viruses upon the body is called *infection*. It is also important for you to remember that all of those diseases which are included under the general head of infectious diseases have their own specific morbid product, which will produce these, and only these, diseases; and although these different diseases may have very many symptoms in common, and may very closely resemble each other in the phenomena which attend their development, yet the specific character of the morbid agent which has produced them stamps them as distinct diseases. There is reason to believe that not one of this class is of spontaneous origin, but that each depends on its own specific poison. As to the exact nature of such a poison, and its element of power in the production of disease, we have no positive knowledge; at the present time, in regard to it, there are two prominent theories.

The *first* is based upon chemical processes ; the *second*, upon the multiplication of living organisms.

The *chemical theory* maintains that after the infectious element has been received into the blood it acts as a ferment, and gives rise to certain morbid processes upon the principle of catalysis.

The *theory of organisms*, or the germ theory, as it is called, maintains that the infectious poisons are living organisms, which, being received into the blood, reproduce themselves indefinitely, and by their reproduction morbid processes are excited which are characteristic of certain types of disease. This is a very seductive theory, and at the present time is quite extensively adopted by medical theorists, as it so readily explains very many remarkable facts connected with the development and reproduction of the class of diseases which are soon to engage our attention. It is readily understood, and there are so many animal poisons which appear to act in this manner, that to one whose opinions are not based upon clinical experience and actual contact with disease, the arguments in its favor seem conclusive.

According to this theory all the different forms of disease included under the head of contagious or infectious may be reduced to, or embraced in, two classes :

First, infectious diseases which depend for their development upon a living *animal* organism. *Second*, those which depend for their production upon a living *vegetable* organism. Unfortunately for this theory, the special organism of any one of the infectious diseases has never been so plainly described by any one competent observer that all others in the same field of study could with certainty recognize it. The bacterian theory, which recently has so occupied the attention of medical men, especially in Germany, is rapidly being disproved, and consequently as rapidly being abandoned. In this country it can scarcely be held to have ever gained a foothold. It seems to me that one who has watched bacterian development must arrive at the conclusion that bacteria found in connection with the development of disease are the product and not the cause of

the diseased process; certain it is that the theory that there exists distinct typhoid, typhus, and diphtheritic living germs, which are the propagating element of these different diseases, still lacks that proof which will lead the practical physician to adopt it. The question then comes back to us, what is the real nature of those morbid substances which, when received into the human organism, have the power of manifesting phenomena which characterize that class of disease which we term infectious? Every day's experience must convince the careful observer that each one of this class of diseases has a distinct producing cause—that the poison of typhus will not produce typhoid fever, neither will the poison of measles develop scarlatina. Although the phenomena which attend the development of these differing diseases may have many points of resemblance, yet each has a distinct origin, that is, has its own specific infection, which specific morbid substance, whenever introduced into the animal economy, either through the skin, respiratory organs, or digestive surfaces, interferes in a greater or less degree with the functions of organic life. This interference is caused either by changes which it produces in the constituents of the blood, or in the solid organs and tissues to which it is conveyed by the blood-vessels and lymphatics.

After reviewing these differing theories and giving careful attention to the facts presented in their support, we arrive at this conclusion—that the exact nature of these morbid agents is unknown. We know that they exist, from the diseased action which they produce; and from the manner in which these diseases are propagated we decide that their poisons are distinct from all other poisons, and that each is specific and can reproduce itself to an unlimited extent. The germ theory best explains the phenomena of development. The chemical theory has decided claims on our acceptance; but until our explorations shall have been carried so far as to determine, beyond question, what is the exact nature of several of these poisons, we shall be compelled to call them unknown morbid agents, governed by certain fixed laws of development and propagation. At

the present time investigation in this direction has scarcely begun.

As we pass from the general causation of this group of diseases to their classification, we find ourselves still in doubt. The symptomatic basis of classification of the earlier writers gave place to the more scientific and comprehensive anatomical basis of classification. This for a long period has been almost universally adopted, yet now is giving place to the recent and more definite etiological classification of the present day.

When these diseases are classified upon an etiological basis, very naturally they divide themselves into three classes.

First.—A class in which the morbid agent cannot be developed exterior to a living being, but, when developed within the system of one individual, can be transferred to another through the atmosphere. Such is the case in measles, small-pox, and typhus fever.

Second.—We have another class called miasmatic or malarial diseases, in which the morbid agent is developed exterior to a physical organization, and cannot be conveyed from one individual to another.

Third.—There is a class in which the morbid agent is developed within, and reproduced exterior to a physical organization. In this class, the poison is developed within the body, but in order that it may be reproduced it must be deposited in decomposing organic matter exterior to the body; it is then rapidly reproduced, and when received into a healthy organism gives rise to diseased processes. It cannot be directly conveyed from the sick to the healthy, but only through the excrements of the sick, or through decomposing organic matter exterior to the body, with which such excrements must have been in contact. There may be all the elements necessary to its reproduction, such as decomposing animal and vegetable matter, but the disease will not be developed unless there has been added to this decomposing mass the specific poison of the disease.

The diseases thus developed have been called *miasmatic-contagious*, of which typhoid fever is the best example.

All the different forms of acute contagious-miasmatic or miasmatic-contagious disease may be either *endemic* or *epidemic*.

They are *epidemic* when they attack a large number of persons at the same time and in the same manner.

They are *endemic* when they are often repeated in the same locality. If they attack individuals without regard to time and place, they are called sporadic.

With this brief introduction, we will enter upon the study of that class of diseases which during the present century have been included under the general head of *fevers*.

Adopting an etiological basis of classification, I shall divide fevers into three classes.

First. CONTAGIOUS FEVERS.—I shall include under this head all those fevers which depend for their development on a specific morbid agent, which agent must originate in an individual suffering from a like specific disease.

Second. MIASMATIC OR MALARIAL FEVERS.—I shall include under this heading all those fevers which depend for their development on a morbid agent developed exterior to the body, and not connected with any previously diseased physical organization.

Third. MIASMATIC-CONTAGIOUS FEVERS.—I shall include under this head those fevers which depend upon a morbid agent developed exterior to the body in animal and vegetable decompositions, to which has been added the specific poison of the fever which has had its origin in a diseased physical organization.

The following is the classification which I shall adopt :

CLASSIFICATION OF FEVERS.

First Class.—Contagious.

TYPHUS FEVER,
SMALL-POX,
MEASLES,

RELAPSING FEVER,
SCARLET FEVER,
MILIARY FEVER.

Second Class.—Malarial.

SIMPLE INTERMITTENT FEVER,	SIMPLE REMITTENT FEVER,
PERNICIOUS FEVER,	DENGUE FEVER,
TYPHO-MALARIAL FEVER.	

Third Class.—Miasmatic-Contagious.

TYPHOID FEVER,	YELLOW FEVER.
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The third class of fevers is a connecting link between the first and second class.

In their pathology and clinical histories the fevers of this class have many things in common with those of each of the other classes, as also in their origin, nature of poison, etc. On this account, and from the fact that during the course of every fever some of the phenomena of typhoid fever are presented, I shall first describe those fevers included in the third class, and shall commence with *typhoid fever*.

TYPHOID FEVER.

This is the most universally prevalent of all fevers. So far as we know, there is no place where it may not be developed and spread. It more frequently prevails in the temperate zone than in the torrid or frigid, but it is possible for it to be developed in all latitudes and in all countries.

This disease, which is essentially the same in all countries, is designated by different names. American writers describe it under the name of *typhoid fever*. The French call it the *typhoid affection*, or *dothi-enteria*. English writers describe the same form of disease under the head of *enteric fever*. The Germans call it *abdominal typhus*, or *gastric fever*. I prefer the name typhoid fever, and will commence its history by describing its anatomical lesions.

MORBID ANATOMY.—As soon as the disease is fully established a change in the blood occurs. It becomes darker in color, coagulating imperfectly, the serum being imperfectly separated from the solid constituents, and is of an unnaturally yellow color. The question arises—did these changes take place in the blood prior to the occurrence of the fever, between the exposure and the period of attack? It is cer-

tain that as soon as the characteristic symptoms of the disease are present, the diminution in the fibrin of the blood is in exact proportion to the severity of the fever, and the number of white globules is increased in a similar ratio.

As a consequence of these blood changes, or in connection with them, a series of changes takes place in those organs and tissues of the body in which the processes of waste and repair are most rapidly going on. These changes are of the nature of parenchymatous degeneration—the essential constituents of the affected organs and tissues being involved.

Similar parenchymatous changes are met with not only in typhoid fever, but to a greater or less extent are characteristic of other fevers and acute infectious diseases.

SPLEEN.—The organ in which parenchymatous degeneration occurs earliest and most extensively is the spleen.

We find this organ undergoing three distinct changes.

First.—It is increased in size, sometimes enormously. The enlargement commences soon after the beginning of the disease, and goes on rapidly until the third week, after which it ceases, and after a few days the spleen begins to diminish in size. If recovery takes place, by the time it is reached the spleen will have returned to its normal size.

The splenic enlargement is apparently due to congestion and to an increase of normal elements.

Second.—As soon as the spleen reaches its maximum size, its consistency becomes soft; this softening is sometimes so marked that, if a post-mortem be made at the end of the third week, the spleen will present the appearance of a dark, jelly-like mass, which is easily broken down.

Third.—The organ becomes almost black in color, owing to the intense congestion which attends its enlargement, and to the deposit of a brown pigment in its substance.

These changes in the spleen take place, in a greater or less degree, in ninety-eight cases out of every hundred.

At the post-mortem of those who have died of typhoid fever, infarctions are sometimes found, although there is nothing peculiar about them. In rare instances, rupture of the spleen occurs without infarctions.

LIVER.—Changes in the liver are by no means as common

as those in the spleen. The liver may be found presenting its normal appearance, or it may be soft and flabby. When soft and flabby, a microscopic examination shows the liver cells more or less granular and fatty, the nuclei of the cells can no longer be seen, and the degeneration may become so extensive that the outline of the hepatic cells is lost, and nothing but a mass of granules remain.

Occasionally there will be found in the liver of those who have died of typhoid fever small grayish nodules situated along the course of the small veins; these nodules consist of lymphoid cells.

The lining membrane of the gall-bladder sometimes presents evidences of catarrhal or diphtheritic inflammation, when there has been no evidence of its existence during life; cases are recorded where it has been found ulcerated.

KIDNEYS.—Degenerative changes in the kidneys are of not infrequent occurrence in the course of typhoid fever; they vary in extent with the duration and severity of the fever. When present, they are more marked in the cortical than in the medullary portion of the organ. In some cases they are confined to the epithelial elements, while in other cases degeneration of all the anatomical elements of the organs can be found. Such extensive changes are less liable to occur in typhoid than in typhus fever. Small gray nodules similar to those referred to as occurring in the liver are sometimes found.

If the epithelial degeneration of the cortical substance is extensive, the cells finally break down into a granular detritus, and the cut surface assumes a yellow color and is softer than normal. Infarctions are sometimes met with in the kidneys of those dying of typhoid fever.

HEART.—The parenchymatous changes which take place in the heart are more marked than those in any other organ, for its anatomical elements undergo waste and repair more actively than those of any other organ; and if faulty nutrition is an important element in these degenerative changes, this organ must become very markedly involved.

In a large proportion of cases it becomes soft and flabby, and is of a grayish or brown color. Sometimes it is so much

changed that its tissues are easily broken down by moderate pressure; it loses its normal outline, and when removed from the body the walls of its cavities readily fall together. When its muscular tissue is examined microscopically, in many instances it will be found that granular changes, affecting the ultimate muscular fibres, have occurred; this granular muscular degeneration may involve a large portion of the organ, or it may be confined to a few muscular fibres. It may be a general or a localized parenchymatous degeneration. Occasionally the muscular fibres are infiltrated with brown pigment.

If, as is sometimes the case, the heart retains its normal outline, is friable, and its cut surface glistens, the muscular fibres will be found to have undergone a change which closely resembles amyloid degeneration; the muscular fibres will be filled with a material which presents the same shining appearance as the amyloid substance, but on applying the iodine test the same reaction does not take place. It is a form of degeneration which occurs in typhoid fever and is not confined to the muscular tissue of the heart, but is found to a greater or less extent in the voluntary muscles of the body.

Thrombi are sometimes found in the heart, and vegetations adhering to the valves and chordæ tendineæ. These may give rise to infarctions in the different organs of the body. The existence of the degenerative changes in the heart, to which I have referred, may be recognized during the life of the patient, for the heart sounds become feeble according to the extent of the degeneration. In some cases the first sound of the heart will be absent, and it has been claimed that when this phenomenon is present the use of stimulants in large quantities is indicated.

LUNGS.—The lungs undergo changes which have received the name of splenization. This is a form of pulmonary congestion which has received its name from the close resemblance which the affected portion of lung tissue bears to the spleen.

The affected lung tissue is of a darker color than normal, and scattered through its substance will be seen little red

or yellowish white points; these little points are scanty blood extravasations.

Lung tissue in a condition of splenization is of a dark reddish blue, brown, or black color; its consistency is firmer than normal, crepitates less freely, has a more uniform, homogeneous appearance upon its cut surface, and is less moist than normal lung tissue; a dark fluid will sometimes ooze from its cut surface, but not as freely as in hyperæmia, and the fluid is more watery in appearance.

A microscopical examination of lung tissue in this condition shows the capillary vessels filled with blood, and the alveoli containing a variable number of cells. In other words, it is a condition closely resembling that condition known as static pneumonia, but no inflammatory process exists; it is simply a stasis in the capillary circulation, accompanied by a slight increase in the cell elements in the alveoli.

BRONCHIAL TUBES.—You will rarely make an autopsy upon one who has died of typhoid fever, without finding evidences of a more or less extensive catarrhal inflammation affecting the bronchial tubes. So constantly is catarrhal bronchitis present in this fever, that Dr. Stokes proposed to call typhoid fever bronchial typhus. In most cases this catarrh is not extensive, affecting only the larger bronchi; it may, however, extend to the smaller tubes and give rise to capillary bronchitis and broncho-pneumonia. Pulmonary infarctions are frequently found in the lungs of those who have died of typhoid fever. They are sometimes quite numerous, are usually of small size, and vary in appearance according to the stage of their development. When recent they are of dark color, and feel like consolidated lung tissue; later, the color changes to yellow; they may soften and break down.

LARYNX.—The larynx, as well as the bronchial tubes, is frequently the seat of catarrhal inflammation; less frequently it is the seat of diphtheritic inflammation. In connection with these laryngeal inflammations, ulcers appear in the larynx; these have received the name of "typhoid ulcers of the larynx;" sometimes they give rise to quite

extensive hemorrhages. In connection with, or independent of these laryngeal ulcers, ulceration of the mucous membrane of the mouth and pharynx may occur; at times it involves the epiglottis in such a manner as to clip off its edges. These ulcers may develop on the mucous membrane of the Eustachian tube. In those cases where permanent deafness follows an attack of typhoid fever, it will usually be found due to ulceration of the mucous membrane of the Eustachian tube.

BRAIN AND NERVOUS SYSTEM.—As yet we have not been able to determine whether there are any structural changes in the brain or nervous system so constant that they may be regarded as lesions of typhoid fever, although it is reasonable to infer that in a disease where such severe functional disturbances of the cerebro-spinal system exist there must be constant and definite parenchymatous changes. Œdema of the pia mater and of the brain substance, with occasionally quite extensive adhesions of the dura mater to the cranium, not infrequently exist. Punctate extravasations into brain substance are found in a certain number of cases, but even in severe cases they are not always present.

STOMACH.—The changes which occur in the stomach are equally important with those that occur in the other internal organs, and are degenerative in their nature. Softening and degeneration of its glandular structure is sometimes so extensive, that if recovery from the fever takes place, a very long time must elapse before the organ can perform its normal function. It is the existence of these degenerative changes that gives rise to the disturbance in digestion which is present in so many cases, not only during the continuance of the fever, but during convalescence.

MUSCLES.—In addition to the degenerative changes which I have described as occurring in the internal organs in typhoid fever, I must say a word concerning those which so recently have been found almost invariably present in the voluntary muscles. This muscular degeneration is of two varieties: First, a granular degeneration, which corresponds to ordinary fatty degeneration. Second, a waxy degeneration, which consists in the conversion of the

contractile substance of the primitive bundles into a homogeneous, waxy shining mass. Often both forms of degeneration occur together, sometimes one and sometimes the other predominating.

In both forms of degeneration the muscular fibres become thicker and more brittle than normal. In the highest degrees of degeneration the muscular fibres are entirely lost, and the muscle may present a yellowish or whitish appearance, so that hardly any traces of the normal color of the muscle remains. During convalescence the normal red color of the muscle returns. This muscular degeneration, however, is not peculiar to typhoid fever, but is met with in all severe infectious diseases.

The want of muscular power, which is so prominent a symptom during the height of the fever, may depend on the disturbances of the nervous system, but the excessive loss of muscular power which is so often present during convalescence is due almost entirely to the muscular changes. The physical strength returns gradually during convalescence as the muscles are regenerated, and it may be months before it is fully re-established. The muscles of the tongue undergo degeneration in the same way as the other voluntary muscles, which accounts in some degree for the interference with the function of that organ so often a prominent phenomenon of the disease.

The *salivary glands* enlarge, become firm and tense, and assume a more or less brown-yellow color. They have the consistency of cartilage. Late in the disease the hardness diminishes, and they assume a red color. These changes are due to a parenchymatous degeneration of the glands, which has been preceded by a cellular hyperplasia. It accounts to a certain extent for the diminution of the salivary secretion, causing a dryness of the patient's mouth, which is so marked and constant an attendant of the fever.

Similar cellular and parenchymatous changes take place in the pancreas.

Changes similar to these occur in other febrile diseases, so that they cannot be regarded as characteristic of typhoid fever.

LECTURE II.

TYPHOID FEVER.

Morbid Anatomy (continued).—Intestinal Lesions.—Etiology.

AT my last lecture I completed the history of those parenchymatous changes which are most frequently met with in typhoid fever. I mentioned that these changes could not be regarded as characteristic of this type of fever, for they are present in other diseases. By some these degenerations are regarded as the necessary result of a prolonged high temperature, but they are in no way different from those degenerations which occur as the result of blood-poisoning where prolonged high temperature does not occur. Especially is this the case in those diseases which are marked by their malignity rather than by their high temperature, as, for instance, acute yellow atrophy of the liver.

Continuing the history of the morbid anatomy of this fever, I now come to those changes which occur in the lymphatic system of the intestinal track.

THE INTESTINAL LESIONS.—These are the most important pathological lesions, and have been called the characteristic lesions of the disease, as these intestinal changes distinguish this fever from all other forms of acute disease.

As the poison of small-pox manifests itself by certain changes in the tegumentary investment of the body, and the poison of epidemic cerebro-spinal meningitis by the formation of pus in the meshes of the pia mater, so the poi-

son of typhoid fever acts directly upon the mucous membrane of the small intestine, giving rise to a catarrhal inflammation accompanied by changes in its anatomical structure, which, in the order of their development, are characteristic of the disease. The character and extent of these changes depend upon the duration of the fever and their nearness to the ileo-cæcal valve; the changes are most marked in the patches nearest to the valve, and less marked in those farthest removed from the valve.

In describing these intestinal lesions, I will suppose that we are examining a severe, well-developed case, which runs its regular course without complication. The changes can be most conveniently studied by *first* considering those which occur within the first week of the disease; *then*, those which are developed within the second week; *next*, those which are most commonly found in the third week; and *lastly* those which occur within the fourth week. They appear to begin as a catarrhal inflammation of the mucous membrane. During the *first week* the mucous membrane surrounding the glands, especially that surrounding the Peyerian patches, becomes hyperæmic and swollen; gradually the glands become more and more elevated, their surface assumes a dark reddish color, interlaced by white lines; this is known as the "*shaven-beard appearance*." These changes begin and are most marked in the glands nearest the ileo-cæcal valve; they are generally well marked within forty-eight hours after the commencement of the disease, but are not fully developed until the end of the first week. By the end of the first week all the glands are involved which are likely to undergo change.

In the *second week*, the mucous membrane of the intestine becomes less red; the agminated and the solitary glands more elevated; the white lines upon their surface disappear, and they assume a uniformly red color. An unusually rapid cell development takes place in the follicles. By this excessive development and the multiplication of the cell elements of this gland structure, the follicles become swollen in all directions. Usually the new cell growth extends beyond the limit of the follicles, so that the

adjoining mucous membrane is also infiltrated with cells. These newly formed cells may wander through the muscular coat and penetrate the sub-serous tissue. By the middle or latter part of the second week the process passes into its second stage, and necrotic changes are established in the newly formed tissue. These morbid changes may terminate in two ways: first, the new elements in these ductless glands may become disintegrated and undergo absorption, and in this way they may gradually undergo resolution; second, individual follicles of the agminated glands may rupture and discharge their contents into the intestine; third, the most frequent and characteristic termination of the typhoid process is the separation of the dead tissue as a slough, and the formation of the typhoid ulcer. Usually the sloughing and removal of the necrotic tissue does not take place until the *third week* of the disease. The surface of the ulcers now presents a yellow appearance, simply because they have been stained yellow by the bile. As the sloughs gradually loosen and fall off, there is a loss of substance which extends to the deeper layer of the mucous membrane, removing the entire gland and the mucous tissue surrounding it, laying bare the muscular coat of the intestine. The necrotic process may extend and involve the muscular tissue and end in perforation of the peritoneal covering.

The size and form of the ulceration corresponds to that of the necrotic tissue; if an entire Peyerian patch is necrotic, an elliptical ulcer is formed, with its long axis corresponding to that of the intestine. In the jejunum and large intestines the ulcers are usually small and round. The edges of the ulcer are sharp, tumid, and overhang the floor of the ulcer. Sometimes the ulcers are hemorrhagic.

In the *fourth week* the process of cicatrization is commenced. Gradually the swollen edges of the ulcers subside, granulation-tissue springs up from their base, connective-tissue membrane is formed, the edges of the ulcers become united at their base, which is covered with a layer of epithelium. The gland structure is never regenerated. The cicatrix which is formed by the healing of these ulcers

is slightly depressed, and less vascular than the surrounding mucous membrane. During the healing process the cicatrix becomes more or less pigmented; these pigmented scars may be recognized years after the cicatrization has taken place. These cicatrices seldom cause any puckering or diminution in the calibre of the intestine. In many cases the process of cicatrization does not pursue this regular course; while one portion of the ulcer is cicatrizing, the process of ulceration in another part may be extending; such long-continued ulceration may prolong convalescence, and even cause death from exhaustion.

I will now briefly review these intestinal changes, and if you will bear in mind the weekly order in which they occur, you will better remember them.

The first thing noticed is congestion, which is most marked around the glands; with this congestion the glands become changed in color. Next, the glands become enlarged, which enlargement is due to a rapid development of cells within their structure; these cells are for the most part lymphoid; but, in addition, there are present large, round cells, with several nuclei. These large, round cells are formed not only in the glands, but in the mucous and submucous tissue adjacent to them; consequently, the enlargement encroaches more or less upon the surrounding mucous membrane. These newly formed cells not only swell the glands and press upon each other, but they press upon the capillary vessels which furnish these structures with nutrition; consequently, there is an interference of the circulation of the gland structure, and as a result the glands become more or less anæmic; degenerative changes occur as the result of impaired nutrition.

In some of the enlarged glands the new elements become disintegrated and are absorbed, and the process ends in resolution; in others, individual follicles soften, break down, and their contents are discharged into the intestinal canal, and the patches acquire a reticulated appearance. More frequently, a necrotic process is established which causes the removal of the entire gland and its contents, leaving an ulcer with everted and perhaps overhanging

edges, with the muscular coat of the intestine for its base. It is now ready for the cicatrizing process, and if it progresses regularly, first the edge of the ulcer becomes inverted, then the base of the ulcer is covered with new connective-tissue cells, the edges become adherent to it, new connective-tissue cells are thrown out upon the edges, and the formation of new tissue goes on increasing until finally the process of repair is complete. These ulcers do not always run such a regular course and terminate thus favorably. If the nutrition of the glands and the surrounding tissues is so interfered with that a gangrenous ulceration is established, sloughing follows, and the gland, with the muscular and other tissues in the neighborhood of the ulcers which are the seat of cellular infiltration, is removed. In some instances the necrotic process continues to extend and involves the peritoneum, causing perforation of the intestines and a fatal peritonitis. These ulcers may assume a hemorrhagic character, with a surface of a dark color. Under these circumstances they are frequently the seat of profuse hemorrhages, which may destroy the life of the patient. Usually, when such accidents occur, vessels of considerable size are involved in the ulcerative process. Whenever the sloughing process is arrested, repair takes place in the manner already described.

As I have stated, these ulcers may be developed in the jejunum, the ileum, the stomach, and the large intestines. In the lower part of the ileum, at the ileo-cæcal valve, they are usually of large size—so large that only small portions of healthy mucous membrane are left between them; in the jejunum, stomach, and large intestines they are usually round and of small size.

MESENTERIC GLANDS.—Associated with these intestinal changes, analogous changes take place in the mesenteric glands. These mesenteric changes are also most marked in the glands situated nearest the ileo-cæcal valve; they are secondary to the changes in the intestinal glands, and are usually affected in a degree corresponding to the extent of the intestinal lesions. The glands are first congested, then there is a production of lymphoid and large cells similar to

those which are found in the enlarged intestinal follicles, the glands become enlarged, and are the seat of an acute cellular hyperplasia. When the enlargement has attained its full size, the hyperemia diminishes, and the cellular elements begin to disintegrate and are absorbed. In about one-half the cases the enlargement reaches its maximum size by the middle of the second or at the commencement of the third week. The enlarged glands vary in size from that of a hazelnut to a small hen's egg. In the stage of retrogression some of the glands simply shrink and return to their normal condition: in other glands partial softening takes place and afterwards absorption, leaving a fibrous cicatrix. If the glands reach a very large size, absorption is incomplete, and dry, yellow, cheesy masses are left, in which after a time salts of lime are deposited and they become enclosed in a fibrous capsule. In rare instances the glands become fluid, their capsules are destroyed, and the softened masses escape into the peritoneal cavity and cause peritonitis.

A calcareous condition of the mesenteric glands, like the pigmented cicatrices of the solitary and agminated glands, give evidence of a previous severe attack of typhoid fever. There is yet another pathological lesion of typhoid fever occurring during convalescence, concerning which I will speak—*namely*, a suppurative inflammation in the cellular tissue upon the surface of the body. The inflammation is not of an active type, but is accompanied by some redness and pain; gradually a tumor is formed at the seat of the inflammation; usually this occurs where there is the greatest amount of pressure. After a time fluctuation becomes distinct, the swelling increases: sometimes two or more of these swellings coalesce, and finally an immense abscess may be formed, which when opened will discharge a pint or more of pus. These abscesses are due to suppurative inflammation in the cellular tissue of the skin. Retro-pharyngeal ulcers are the result of suppurative inflammation of the connective tissue. As a result of imperfect nutrition of the skin, a gangrenous inflammation of the skin may be developed, which gives rise to "bed-sores," as they are

commonly called. These are especially liable to occur in the latter stages of a typhoid fever which has been attended by a prolonged high temperature. The slough may form over the trochanters, over the sacrum, or wherever the tissues have been subjected to pressure for a long time, and is a consequence of impaired nutrition of the skin. Sometimes this gangrenous process not only involves the skin, but also the subjacent cellular tissue and the muscles. Gangrene of the toes and portions of the integument which are not subjected to pressure is due either to thrombosis or embolism.

This completes the history of the anatomical lesions of typhoid fever. In connection with this history I would call your attention to something of special importance, which I wish you would remember, namely, that typhoid fever is a *specific disease*; that it has a specific pathological lesion, a catarrhal inflammation of the intestinal mucous membrane, attended by special follicular changes; and though you may find present in other diseases changes closely resembling those which I have described as the characteristic lesion of typhoid fever, yet there is no other disease in which these changes have a regular development, in which the different stages can be indicated with a degree of certainty by days and weeks.

ETIOLOGY.—We very naturally pass from the consideration of the morbid anatomy of typhoid fever to its etiology. According to the classification of fevers which I have adopted, it is included in the list of miasmatic-contagious fevers. Usually, it has been regarded as an endemic form of disease. There seems to be no connection between its development and destitution; for not only does it choose its victims from the hovels of the poor, but from the dwellings of the middle classes, and from the palaces of the rich. It may occur as an isolated case, or whole households and neighborhoods may be stricken down with the disease. We must therefore regard the causes of its production as local and limited, and not widespread. It is possible for it to prevail as an epidemic, but it must first have been endemic.

In studying the etiology of this fever, two prominent questions present themselves:

First.—Is it a contagious form of disease?

Second.—Is it ever of spontaneous origin?

The question of contagion is one that has been very thoroughly discussed. For many years representative medical men have differed upon this point. After years of careful investigation, I think it may be now unhesitatingly stated that facts do not sustain the opinion that typhoid fever is ever, strictly speaking, a contagious disease, or that it is ever directly transmitted from one individual to another.

Persons sick with this fever are now admitted into our general hospitals, and are placed in beds by the side of patients sick with pneumonia or any form of chronic disease, without endangering the lives of such patients. This fact shows how generally the profession regard this disease as non-contagious. Typhoid fever is no longer restricted by quarantine regulations. All these facts tend to dispose of the question, Is it a contagious disease?

The question, Is typhoid fever of spontaneous origin? has also been thoroughly discussed, and there are strong advocates on both sides of the question.

Some of those who believe that it may have a spontaneous origin maintain that the poison which gives rise to it is developed by the decomposition of organic matter, and that the specific character of the fever is due to the particular substances which are undergoing decomposition. Others maintain that the decomposing substance is mainly human excrement—in other words, that decomposing human excrement is necessary for the production of the peculiar poison which gives rise to typhoid fever. Again, others who believe that the disease is of spontaneous origin maintain that the presence of vegetable matter in certain conditions is necessary for its production, and that these conditions are similar to those which exist when miasmatic fevers are developed, the difference in the two poisons depending rather upon the rate of temperature than upon the character of the ingredients.

There is a view only recently advanced in regard to the origin of typhoid fever, that sewer gases contain the poison which has the power of developing the disease.

On the other hand, it is maintained by those who do not believe in the spontaneous origin of this fever that, in addition to decomposing animal and vegetable matter, it is necessary that the specific typhoid poison shall be incorporated in the decomposing mass. It is the leaven (if you choose so to call it) which is to leaven the whole mass. Daily observation seems to prove clearly that vegetable or animal decomposition alone is not sufficient for the development of this disease, even admitting that it depends upon the decomposition of human excrement. In how few of the many dwellings permeated with the effluvia from privies do the inmates have typhoid fever.

Again, facts do not sustain the claim of those who say that sewer gases contain the typhoid poison, for those cities in which the sewerage is most imperfect, and those houses most frequently permeated with sewer gases, are not the hotbeds of typhoid fever. Again, this fever is more prevalent in the country than in the city, in places where there are no sewer gases; indeed, well-marked cases of typhoid fever are of quite rare occurrence in the city, and when they do occur seem to be developed independently of defective sewerage. In other words, all the elements which favor its production may be present, such as animal and vegetable decomposition or sewer gases, and yet not a single case of typhoid fever be developed, until some person having typhoid fever comes within the precinct, or some substance containing the typhoid poison is brought within the boundaries favorable to its development; then a severe endemic of the disease may be developed.

In carefully reviewing the history of the origin of this disease in the different localities in which for the first time it has suddenly appeared and prevailed, we will find that the advent of one suffering from the disease, or the introduction of matters from such a person, has been the starting-point of the endemic; in other words, that one of these two conditions is a primary necessity for its production.

We therefore almost necessarily reach the conclusion that something is necessary for the development of the typhoid poison besides favorable external conditions; that animal

and vegetable decomposition does not primarily originate the poison, but furnishes a favorable soil for its growth and development.

Facts warrant us in making the statement that while on the one hand typhoid fever cannot be regarded as a strictly contagious disease, on the other hand it is not of spontaneous origin.

It is hardly necessary for me to review all the facts which have a bearing upon this subject. I believe any unprejudiced person will arrive at this conclusion from the careful study of them, that when typhoid fever makes its appearance in any locality, its development is preceded by the introduction of a specific typhoid poison, which has been reproduced (in most instances) in connection with decomposing human excrement.

The question now arises, What is the real nature of that poison derived from a person sick with typhoid fever, which has the power of indefinitely reproducing itself outside of the body in connection with decomposing organic matter, and thus becomes the infecting agent, when individuals are brought within its influence?

The history of endemics of typhoid fever leads to the conclusion that the poison is contained in the faecal discharges of the sick. When such excrement is in a fresh condition the poison is not active; it must go through a stage of development outside of the body. This may take place in the excrement itself, but it goes on more rapidly and abundantly if the excrement is collected in privies or in earth that is already saturated with organic matter. In this way you can readily explain how a typhoid fever patient coming into a locality previously free from the disease can establish there a focus of infection, from which many persons may become diseased.

It is evident that this poison is not active in its fresh state, from the fact that the disease is not carried directly from one individual to another—as attendants, nurses, and physicians are no more liable to the disease than those who are in no way exposed to the disease and live in a healthy locality. Mothers may sleep in the same bed with children

who are sick with the fever without contracting the disease. As has already been stated, in order that typhoid excrement shall become effective in the transmission of the poison, it is necessary that it should go through a stage of development in connection with organic matter outside of the body; so it passes from the diseased individual to the localities which are favorable to its development, and again from these localities into the human body.

It is difficult to determine the period of incubation, or length of time the poison must remain in the body before symptoms of the disease are manifest. The history of isolated cases would lead to the conclusion that the period varies from fourteen to twenty days.

The next question that arises is, How does the typhoid poison gain admission to the human body? Undoubtedly there are two principal sources of infection, namely, the air we breathe and the water we drink. A large number of well-authenticated histories have now established the fact, that this fever may be developed by gases which emanate from privies, sewers, etc., which have been the receptacle of excrement from typhoid patients, and also, by drinking water from springs and wells which have become contaminated by matters from adjoining privies and cesspools. It is also now an accepted belief, or rather, is regarded as an established fact, that water remains contaminated, though far remote from the point where it came in contact with a defective sewer or water-closet.

Soil pipes and sewerage may be defective for a long time, perhaps a year, or even longer, and no case of typhoid fever occur, when suddenly an endemic of typhoid fever breaks out, and careful investigation shows that its development was preceded by the introduction of the excrement of a single individual sick with the disease.

It is the belief of some that milk can convey the typhoid poison, and there is evidence in favor of this opinion; but I think there is stronger evidence that the water used to dilute the milk, and not the milk itself, is the medium through which the poison is transmitted.

This poison has great vitality. Typhoid fever frequently

occurs in the same locality year after year, when the surrounding conditions are favorable to its development. Those conditions which favor its development are more frequently present in the autumn than at any other season of the year, and for this reason it has been called Autumnal fever.

Usually it makes its appearance in a locality, year after year, at about the same time: case after case is developed until entire households and neighborhoods become its victims. Individuals who come to care for the sick may contract the disease, and even persons who visit houses in which the disease is prevailing may afterwards develop the fever, contracting it, not from the sick, but from the infected atmosphere of the locality.

Age must be regarded as a predisposing cause of typhoid fever. It is much more likely to occur in young than in old persons; it occurs most frequently between the ages of fifteen and twenty-five, and is rarely met with in persons over fifty.

There are also individual idiosyncrasies which seem to predispose to this fever. Some contract it upon the slightest exposure to the influence of the poison, while others, frequently brought in contact with it through long epidemics, escape. Again, an individual may have repeated attacks of typhoid fever. I have in mind a physician who had typhoid fever four times, the last attack proving fatal. A person who has had typhus or scarlet fever is not likely to have a second attack, but no such immunity follows an attack of typhoid fever. Whatever view we take of the exact nature of the typhoid poison, it has been quite conclusively demonstrated that this poison differs very essentially from that of other fevers.

From this brief review of the etiology of this fever, we are led to the following conclusions:

First.—That its development is independent of overcrowding, and that it attacks the rich and poor indiscriminately.

Second.—That it may be communicated from one person to another through the excrements which have undergone decomposition after their discharge.

Third.—That an endemic of typhoid fever only occurs where the air or drinking water of the locality has become poisoned by emanations from typhoid excrements which have undergone decomposition, and that, if the fever becomes epidemic, it is a circumscribed epidemic, and not widespread.

Fourth.—That the exact nature of the typhoid-fever poison is still unknown.

LECTURE III.

TYPHOID FEVER.

Symptoms.

I SHALL this morning commence the history of the symptoms of typhoid fever.

If I should attempt to give you a correct picture of this disease—one perfect in all its colorings—it would occupy too much time, and you would become so confused as to be unable to recall even the outline of the picture.

After I have briefly spoken of the manner in which this disease makes its advent, I shall consider the prominent symptoms of a typical case, and then discuss in detail these symptoms, without special regard to the time of their occurrence. This fever is usually insidious in its approach, and comes on with a certain degree of uneasiness throughout the system; the patient feels uncomfortable, has no pain, but feels that he is about to be sick. If the individual is in a region where the disease is prevailing, it is quite common to hear the expression, “I believe I am going to have the fever,” and yet those who make such complaint will scarcely admit that they are sick. They complain of a grumbling headache, more or less aching of the limbs, “a tired feeling all over,” chilly sensations, alternating with flashes of heat; loss of appetite, and not unfrequently nausea and vomiting are present. These premonitory symptoms gradually increasing in severity, by the fifth or sixth day the patient is compelled to take to his bed. At this early period there may be a slight diarrhoea. In very mild cases the disease comes on so insidiously, and with symptoms so mild, that the patient is often able to pursue his

ordinary avocations, complaining only of an undefined indisposition—not feeling exactly well, but not regarding himself as really sick. In very many severe cases it is impossible for the patient to accurately fix upon the time when the fever commenced. In no case will you be able to make an early positive diagnosis. Typhoid fever may be suspected, but that is as far as you can safely go.

In all cases variation in temperature is one of the most important early symptoms. Such variation in temperature in a typical case may be divided into four periods, of one week each, which correspond to the four weeks of the disease.

In the first week there is a gradual and steady rise in temperature, with regular morning and evening variations.

This is one of the characteristic features of the disease. If, in any case of fever, you find, while making your thermometrical observations, that there is a gradual rise in temperature, marked during the first week by regular morning and evening variations, you may be quite certain your patient has typhoid fever. This gradual rise of, and these variations in temperature are not present in every case, but when they are present they will greatly assist you in making an early diagnosis.

It has been said that typhoid fever is the only disease, except double quotidian intermittent fever, that gives two full thermometrical curves within twenty-fours; that is, two full remissions and two exacerbations. If this is true, it helps to explain certain high temperatures in the morning, and affords valuable assistance in making a diagnosis.

During the second week the variations in temperature are slight, retaining, however, the same degree of exacerbation which was reached at the end of the first week.

The variations during the third week are remittent in character.

During the fourth week they become intermittent, and the range of temperature in the exacerbations is lower. The variations in pulse correspond to the variations in temperature. During the first week the pulse gradually becomes more and more frequent, and remains at the height

reached at the end of the first week ; throughout the second and third weeks there are distinct morning and evening remissions ; during the fourth week it falls to its normal standard.

On the seventh day, or sometimes between it and the twelfth day, the characteristic eruption appears. About this time the headache abates and more or less somnolence and delirium come on. The delirium at first is slight, and is only observed during the night. Day by day the patient loses flesh and strength, and becomes more and more unconscious, and all the phenomena of the typhoid state are developed, viz., a dry brown tongue, feeble pulse, low muttering delirium, stupor, tremors, subsultus, involuntary evacuations, and the other phenomena of great prostration.

If the disease is to terminate favorably the amendment is usually gradual. The first sign of improvement is a decided remission of the fever. During the first week there is usually some diarrhœa ; in very many instances it is present before the patient seeks the advice of the physician. It may have ceased at the time he seeks such advice.

Such, in brief, are the phenomena which attend the ushering-in and developing stage of an ordinary case of typhoid fever ; they are, however, subject to numerous modifications. Some cases of this fever are mild throughout their entire course ; some are severe at first and mild afterwards ; some are mild at first and severe afterwards ; while others are severe throughout their entire course.

In the detailed study of the prominent phenomena of this disease I shall not attempt to follow the order of their development, for they are subject to so many variations that such a course is impossible.

In our attempt to analyze its principal symptoms I will first notice the changes which take place in the countenance.

THE PHYSIOGNOMY.—As a rule, in the milder cases, the countenance has nothing peculiar in its appearance ; the patient does not even look ill. If the disease is of a severe type, by the second week the countenance assumes a characteristic appearance—there is a pale, olive, leaden look,

the eye becomes dull and the conjunctiva congested, and usually there is a small, rose-colored spot in the centre of the cheeks. The face does not assume the dark mahogany color, as seen in typhus, but in the advanced stage of the fever it has more of the hectic flush of phthisis.

TONGUE.—The tongue will also present certain changes. From the very outset it is covered with a light, white coat, but there is nothing special in its appearance before the end of the first week; then it may become red upon its sides and tips, and show a slight disposition to dryness in its centre. As the disease passes into its second and third weeks, the tongue becomes more heavily coated, the coating becomes brown and dry, and sordes collect upon the teeth and sides of the mouth in sufficient quantities to form crusts. These crusts may become thicker and more abundant as the disease progresses. At any period in the course of the disease the tongue may suddenly clear off, and present a shiny red appearance, "beef-colored," as it has been called. The tongue and lips may become dry, cracked, and fissured. As the sordes are removed from the lips, they will often bleed; and in certain cases, more especially in the severer forms of the disease, the entire mouth and tongue may be covered with dark-colored incrustations. Such incrustations are seen early in connection with those cases where there are extensive blood-changes; when present they are of grave significance.

As soon as convalescence is established the changes in the appearance of the tongue are very marked. One of the first indications of convalescence is a moist condition of the tongue about its edges; gradually its entire surface becomes moist, and by the time convalescence is fully established it is restored to its natural condition. Gastric symptoms are always more or less prominent—loss of appetite is one of the earliest symptoms, and nausea and vomiting are quite common during the first week of the fever. The vomited matters usually consist of a greenish fluid. When vomiting comes on late in the fever, it is due either to sub-acute gastric catarrh, or it is symptomatic of local or general peritonitis. In a large proportion of cases the thirst is

excessive. The lips are parched, and in severe cases crack and bleed. In some cases hemorrhage from the gums occurs.

DIARRHŒA.—Although not invariably present, it is so frequent an attendant of this fever that it is considered one of its characteristic symptoms. It varies with the severity of the attack, the date of its commencement, and its duration. The characteristic typhoid discharges are of a yellowish green color, described in the books under the term of "pea-soup discharges." Sometimes they are of a dark color, resembling coffee-grounds; their reaction is alkaline. In some cases diarrhœa is present at the very outset of the disease, and continues throughout the entire course. In other cases it does not appear until the third week. The second week is the ordinary time for its appearance. When the diarrhœa appears late in the course of the disease, the discharges are more copious than when it appears early. A mild diarrhœa throughout the entire course of the fever is a favorable rather than an unfavorable symptom. In mild cases diarrhœa is sometimes absent.

INTESTINAL HEMORRHAGE.—Intestinal hemorrhage is not an infrequent attendant upon typhoid fever. It occurs in about one in twenty cases, and varies in quantity from a mere trace of blood in the stools to a profuse discharge of from sixteen to eighteen ounces. The slight hemorrhages which sometimes occur early in the disease simply indicate a hemorrhagic tendency, the same as the epistaxis which is very frequently among the early symptoms. In both instances the bleeding comes from the capillaries of the mucous membrane. The more profuse hemorrhages are due to the opening of an artery in some intestinal ulcer. Hemorrhages due to this cause may be sudden and profuse, and may destroy the life of the patient. The usual time for the occurrence of these profuse intestinal hemorrhages is in the latter part of the second and during the third week. These hemorrhages are usually preceded by a sudden fall in temperature, perhaps two or three degrees; if then in a patient severely ill of typhoid fever a sudden fall in temperature occurs during the second or third week,

accompanied by extreme prostration, it is very conclusive evidence that intestinal hemorrhage has occurred, although externally the hemorrhage may not have made its appearance. When intestinal hemorrhage occurs during the second or third week it must always be regarded as a grave symptom ; yet it is not necessarily followed by fatal results.

The blood is usually fluid, rarely clotted ; generally it is of a bright red color, owing to the alkaline condition of the intestinal contents. Copious intestinal hemorrhages are more frequent in severe cases that have been attended by profuse diarrhœa. In one or two instances I have had patients die of intestinal hemorrhage before any blood had been voided externally. If the patient survive a profuse intestinal hemorrhage, there is great danger of his dying from peritonitis. He may die unexpectedly by syncope a number of hours after a profuse intestinal hemorrhage.

Abdominal pain and tenderness are not usually present at the very outset of typhoid fever ; generally, and almost without exception in the severer cases, by the sixth day of the disease some pain and tenderness will be present in the right iliac fossa. The pain and tenderness usually increase as the disease progresses, and in the advanced stages it is sometimes so marked that slight pressure over this region gives the patient great pain. While examining this region in order to determine the presence or absence of pain and tenderness, remember never to press the surface with the ends of the fingers, but always make the examination with the palm of the hand ; while making the pressure watch the face, and frequently you will be able to determine by the expression of countenance whether you are, or are not, causing pain, long before an audible complaint is made by the patient.

It is also important for you to bear in mind the possible occurrence of a more severe abdominal pain—namely, that pain arising from intestinal perforation. The following are the characteristic symptoms of this lesion. If in the course of a slight or severe form of this fever, or even when the disease has been latent and the diagnosis of typhoid fever

has not been clear, the patient should be suddenly seized with diarrhoea, pain in the abdomen, aggravated by pressure, perhaps at first localized in the right iliac fossa, but soon extending over the entire abdominal cavity, attended by symptoms of great prostration, a rapid, feeble pulse, a sunken, anxious expression of countenance, rapid tympanitic extension of the abdomen, nausea and vomiting, quickly followed by coldness and blueness of the extremities, and the other signs of sudden collapse, you may be almost certain that perforation of the intestines has occurred. I have known this accident to occur when convalescence was progressing apparently safely and satisfactorily. Few live more than thirty-six hours after the occurrence of the perforation.

Tympanitis is another very common symptom of typhoid fever. Usually it is not present during the first week, but by the end of the first or the commencement of the second week a fullness of the abdomen will be noticed. As the fever advances, sometimes the distention often becomes extreme; this is due to a collection of gas in the large intestine, developed from some change in the mucous membrane, the exact nature of which we do not fully understand. We only know that sometimes the mucous membrane of this intestine very rapidly secretes gas, or allows it to generate, and that the intestine becomes distended by its accumulation. When once it is developed it remains until convalescence is fully established. It is always an important diagnostic sign of this fever. In connection with the development of the tympanitis, when firm pressure is made over the right iliac fossa, a gurgling sound is produced; but *gurgling* in the right iliac fossa cannot by any means be regarded as a positive symptom of typhoid fever, as it may occur in any disease where there is distention of the abdomen due to accumulation of gas in the intestines. In typhoid fever, so long as the abdomen remains tympanitic, no matter what the temperature and pulse of the patient may be, he is in more or less danger, for it shows that there are intestinal changes still in progress, and that the reparative processes are not complete; this is more especially the case

when the tympanitis has continued from the active period of the disease into the period of convalescence. Therefore, the presence of tympanitis during convalescence is never to be lightly regarded.

These are the most important symptoms which are referable to the alimentary tract, and may be regarded as forming, in connection with the temperature variations, the essential part of the history of this fever.

URINE. — Extended and very careful analyses of the changes in the urine of typhoid fever patients have been frequently made, without giving any very practical results.

Usually during the first two weeks of the fever the urine is diminished in quantity; after the second week it is increased. During the time it is diminished in quantity, its color is dark and its specific gravity is high; when it is increased and convalescence is established, it becomes pale, and its specific gravity is lowered.

The amount of urea excreted daily throughout the active period of the fever is increased. The increase is in proportion to the intensity of the fever, subject in some degree to the quantity and quality of the food taken. It will be greater when large quantities of strong beef-tea are taken, than when the diet consists of milk. So long as the kidneys are able to eliminate the excess of urea, no harm results; but if the quantity exceeds their power of elimination, or if their function of elimination is interfered with, uræmic symptoms will be developed, such as delirium, stupor, and coma.

Albumen in the urine is only of occasional occurrence in the course of typhoid fever. When present the quantity usually is small, and it is only temporarily present. It rarely appears before the third week. Its appearance is often marked by the occurrence of cerebral symptoms. Renal epithelium and casts may or may not be present with the albumen. The *spleen* is often much enlarged, and can be felt through the abdominal wall. The enlargement is greatest in persons under thirty years of age, and during the second week of the fever.

NERVOUS PHENOMENA.—The symptoms referable to the

nervous system are not so prominent in typhoid as in typhus fever; yet there are many cases in which these symptoms play an important part in its history.

One of the most constant of this class of symptoms is headache. In the majority of cases it is one of the ushering-in symptoms of the disease. It is present in mild as well as in severe cases; sometimes it is confined to the forehead and temples, more often it extends over the whole head—not violent, but a dull, heavy pain. It usually increases in severity until the middle period of the disease, certainly until the close of the first week; and generally associated with it there is intolerance of light and conjunctival injection, pain in the back and limbs, and a general aching of the whole body.

Somnolence is another nervous phenomenon present to a greater or less degree in all cases. In mild cases it does not appear until late, and usually is not long-continued. In the severer cases it appears early and continues until convalescence begins; in fatal cases it increases up to the time when the patient passes into a state of coma. It is often interrupted by delirium.

In children this symptom is especially prominent, and is very valuable as a means of diagnosis. For example, if a child complains of feeling sick, without any well-defined pain, upon inquiry you find that he has had little or no sleep for two or three days; gradually he passes into a state of somnolence, which at first is slight, but soon it becomes profound; you may infer that typhoid fever is about to be developed.

Delirium is more frequently present than absent in typhoid fever. The character of the delirium varies; the usual form is known as the "low-muttering" delirium. This form is rather characteristic of this type of fever, and yet in very many cases the delirium may be violent in character, and may become maniacal to such an extent as to require physical restraint. Not unfrequently typhoid fever patients attempt to jump out of a window, or to injure themselves or their attendants in their endeavors to escape from fancied pursuers; or they are seized with the

impression that their attendants are their personal enemies, or that within themselves there is something fearful that must be destroyed.

It is very common for the minds of this class of patients to be occupied with those things which engaged their attention just prior to their illness. They imagine persons who are absent are about them, and not unfrequently call them in the most endearing tones, or denounce them with the most violent epithets.

The delirium rarely comes on until the second week of the fever, and it commences and is most active at night. After it has once appeared it usually continues until convalescence is established, and generally disappears during a sound sleep which attends the early stage of convalescence. The maniacal form of delirium in typhoid fever is usually most marked at night. During the low-muttering delirium, if the patient is asked questions, he will generally answer correctly.

MUSCULAR PROSTRATION AND PARALYSIS.—In all severe cases of typhoid fever muscular prostration is noticeable in the early stages, and increases with the progress of the fever. It is generally most marked during the third week. Where there is marked muscular paralysis, the urine and fæces are passed involuntarily, there is inability to protrude the tongue, and more or less difficulty in deglutition. These symptoms are often attended with difficulty or inability to articulate distinctly. Retention of the urine, occurring early on account of the inability of the bladder to evacuate itself, is a very unfavorable symptom; the same is true of involuntary discharges from the bowels.

MUSCULAR TREMORS.—Tremors of the hands, or tongue, or lips, are most often met with in young subjects, and in those who are addicted to the use of spirits. Severe tremors, unaccompanied by much mental disturbance, often attend extensive intestinal changes.

Spasmodic movements, such as subsultus, hiccough, etc., are observed in the advanced stage of severe cases. Rigid contraction of the muscles of the neck and those of the extremities are also sometimes present in severe cases.

General convulsions are of very rare occurrence, except in very young children, and when they occur have no special significance.

SPECIAL SENSES.—The symptoms referable to the special senses require little more than enumeration.

As regards the *sense of sight*, there is nothing worthy of note, except that the eye assumes a dull expression and that the pupil is dilated; some patients complain of haziness of vision, which is increased when they assume a sitting posture.

The *sense of hearing* is always more or less impaired; this is most marked about the middle period of the fever; then it is impossible for your patient to hear ordinary conversation—you will be obliged almost to shout in his ear.

Ringling and buzzing sounds in the ears are often complained of in the early stage of the fever.

When the loss of hearing is confined to one ear, it is generally caused by ulceration of the mucous lining of the Eustachian tube, or by suppuration of the middle ear.

The *sense of taste* usually is altered or perverted; articles of food are tasteless, or have an unnatural flavor. When the tongue and mouth are covered with a heavy coating of sordes, with a tremulous tongue, the patient is unable to distinguish between bitter and sweet, and swallows the most disgusting doses without complaint.

Hyperæsthesia is another disturbance of a special sense. The surface of the body of a typhoid fever patient may become so sensitive that he will cry out with pain from the slightest touch. This hyperæsthesia may be present during the first week, or may not be present until convalescence is established. It is most marked over the abdomen and lower extremities, and usually occurs in females of a hysterical tendency. It is of importance that you discriminate between cutaneous tenderness in the abdominal region, and the tenderness of peritoneal inflammation.

EPISTAXIS.—I have already referred to this symptom as of common occurrence in the early stage of typhoid fever. When it occurs during the first week, in most cases it is of little importance, except as a diagnostic sign of this type of

fever; when it occurs during the third week, it becomes important as an element of prognosis, as it may be sufficiently profuse to destroy the life of the patient. Occurring late in the disease, unless it can be promptly arrested, it always jeopardizes the life of the patient.

Emaciation is perhaps more marked and rapid in this than in any other form of fever. It commences early and is progressive. By the time a patient has reached the fourth week of a typhoid fever of even moderate severity, he is usually in a condition of extreme emaciation. In this particular he markedly differs from a patient ill with typhus fever, for in the latter case emaciation to any great extent does not occur.

LECTURE IV.

TYPHOID FEVER.

Symptoms (continued).—Differential Diagnosis.

I WILL continue the history of typhoid fever, and describe more in detail those *variations in temperature* which attend its development and mark its progress. As has already been stated, the temperature at the commencement of a typical case of this fever is characterized by morning remissions and evening exacerbations; and by these regular variations often you will be able to make a diagnosis during the first week of the disease. In order to estimate the real value of these variations, it will be found convenient to divide the fever into four periods which shall correspond to the four weeks of the disease.

In making your thermometrical observation, in this as well as in all other forms of fever, the thermometer may be placed in the axillæ, the mouth, or the rectum. You must remember, however, that the temperature ranges about one degree higher in the mouth and rectum than in the axillæ. I shall refer to axillary temperature whenever I speak of temperature without qualification.

Usually the temperature begins to rise about noon on the first day of the development of the fever, and continues so to do until between six and eight o'clock in the evening, when it reaches its maximum height for that day; then there is no change until midnight, when it begins to decline, and by six or eight o'clock in the morning it has reached its minimum decline, which is a degree higher than on the morning of the preceding day. After six or eight o'clock in the morning the temperature does not vary much until

noon; then it again begins to rise, and by six o'clock in the evening it has reached its maximum elevation for that day, which is two degrees higher than on the evening of the preceding day. Again, at midnight it begins to fall, and by morning it has fallen a degree, which leaves the maximum temperature for the day a degree higher than on the preceding day. Thus it rises a degree each day, with regular morning and evening variations, until the eighth day of the fever, when, in most cases, it has reached its maximum height. During the *second week* the temperature remains at about the same maximum degree which it has reached by the end of the first week. There are morning and evening variations of a degree or more, but the maximum of the evening exacerbation remains the same.

During the *third week* the remission becomes more and more marked, and with it the temperature falls, while during the exacerbation the temperature retains the same standard as during the second week. By the end of the third week the morning temperature during the remission will have fallen two or three degrees below the point which it had reached during the second week.

By the time the *fourth week* is reached, or at least by the middle of the week, the temperature becomes intermittent, and with each exacerbation it falls lower and lower, until by the end of the week the normal standard of temperature has been reached—it may fall a little below the normal standard.

These are called the typical thermometrical variations of typhoid fever, yet they are not always present; besides, there are many things which will materially modify them. For instance, marked deviations from the record may be produced by complications which would never have been discovered but for the irregular thermometrical variations. By treatment, for a time the temperature can be very much lowered; but, if the treatment be discontinued, it will again rise. In some cases you will be unable to ascertain the cause of the irregularity.

PULSE.—The pulse is also subject to variations, which correspond very nearly with the variations in temperature,

and occur not only on different days, but at different hours on the same day. During the first week the pulse becomes more and more frequent, during the second and third weeks it remains at its height, and during the fourth week sinks to its normal average. During the whole course of the disease it is less frequent in the morning than in the evening.

If, at the commencement of the fever, the pulse is ninety-eight, it gradually increases in frequency, until, by the end of the first week, it has reached one hundred, or one hundred and ten per minute; during the second week it remains at about this height; after that time it may run as high as one hundred and twenty or one hundred and forty. During the first and second weeks the rate of the pulse and the temperature range correspond, but after this time the parallelism ceases, the failure of heart-power beginning to manifest itself. This failure of heart-power is indicated by an increase in the frequency and feebleness of the pulse, which at this time may reach one hundred and forty per minute, and yet the temperature show no alarming variation. A pulse which remains for five or six consecutive days above one hundred and twenty per minute is a bad omen, for it shows extensive changes in the muscular tissue of the heart. Under these circumstances, the pulse may become irregular and intermitting. Should these irregularities and intermissions occur during the third week, in most cases they are followed by death. With an irregular and intermitting pulse, usually, you will find the first sound of the heart inaudible over the precordial space, and this indicates that prompt and judicious means must be employed to restore, if possible, the heart's normal action, and thus relieve your patient and avert a fatal issue.

The severity of the fever during the first and second weeks of its development is, to a great extent, determined by the frequency of the pulse and the height of the temperature. Although delirium and extensive tympanitis are important symptoms, yet they do not determine the result; but if your patient, during the first, or at the commencement of the second week of the disease, has a pulse of one hundred and twenty per minute, and a temperature of one

hundred and six, it is very doubtful whether convalescence can ever be established.

You must remember that from feeble heart-power alone the pulse may increase in frequency, while the temperature is steadily falling. On the other hand, the pulse sometimes falls almost to a normal standard, while the temperature remains high. In either case, if these changes occur during the second or third week of the fever, they must be regarded with suspicion.

ERUPTION.—We now come to the study of what is known as the “characteristic symptom” of typhoid fever, namely, the eruption. Some have claimed that the eruption should be considered as a lesion of the disease, but I prefer to class it among the symptoms. It makes its appearance between the sixth and twelfth days, dating from the commencement of the fever (not from the day the patient takes his bed, but from the time the first symptoms of the disease manifest themselves), and it is not attended by any unusual sensation.

It remains visible from eight to fourteen days, leaving no stain or mark on the surface after its disappearance. It consists of isolated, lenticular spots scattered more or less abundantly over the surface of any part of the body, yet usually most abundant upon the chest and abdomen. There may be only a few spots visible at a time, or it may be so profuse as to cover the body like a rash. Two or three well-defined spots of the eruption are sufficient to establish the existence of the fever. Each spot is circular in shape, and varies in diameter from a point to a line and a half, rarely reaching two lines. It is slightly elevated above the surface of the surrounding cuticle, is of a bright rose color, disappears upon slight pressure, and returns as soon as the pressure is removed. Each spot remains visible for three days, and then disappears. Sometimes, as one crop of the eruption disappears another is developed, and this may go on for eight, twelve, or fourteen days. There are many cases in which only one crop appears. As soon as one spot makes its appearance, it is well to mark it with tincture of iodine or nitrate of silver, so that you may be certain that

your observations are always made upon the one point. If it is a spot of typhoid eruption, and one crop of eruption is to follow another, it will disappear within three days from the time at which it was first seen, and other spots will take its place. It is this feature which distinguishes the typhoid eruption from that of all other fevers.

The question may be asked, Is this eruption essential to the diagnosis of typhoid fever? Doubtless there is no question in connection with its history which has given rise to more discussion than this. As a matter of course, this question has two sides. Many observers mention that the eruption is not constant, and consequently not necessary for its diagnosis; while others, equally competent, maintain that, unless the eruption be present at some period during the progress of the disease, the diagnosis of typhoid fever cannot be made with positiveness. Jenner states that he found the eruption present in one hundred and forty-eight out of one hundred and fifty-two cases. I would not say that it is possible for typhoid fever to occur without the eruption, neither would I affirm that scarlet fever ever exists without the characteristic rash of the disease; but I do say that, as regards these respective fevers, that if no eruption was present, I would make the diagnosis with equal hesitancy in the one case as in the other.

The eruption is usually most marked in cases of typhoid fever which occur between the ages of ten and thirty. Before ten and after thirty years it is usually not as well marked, and may be readily overlooked unless careful search is made.

I have described to you the prominent symptoms which are present during the course of a typical case of typhoid fever, and believe you will now be able to recognize the disease and to manage intelligently your typhoid fever patients.

At this point let me state to you that the typhoid poison, in its operation on the human body, does not always effect the series of changes and symptoms which I have been describing. On the contrary, there are cases which run so mild a course that they can scarcely be dignified by the

name of fever; besides, there are imperfectly developed cases which show a great diversity in their course, but they all can be included under two heads:

First.—*Mild typhoid fever*, in which the symptoms are all mild.

Second.—*Abortive typhoid fever*, in which the duration of the disease is markedly shortened.

In the *mild type*, the fever runs its regular course, but it is of low grade. The temperature rises regularly until its maximum is reached, which rarely exceeds 103° F.; then it remains stationary for a time, generally about a week; then a decline follows in the same manner as was noticed in the typical case. This is the regular course of these cases if left to themselves, and, as a rule, they should be left to themselves. Some of these cases are so mild that the patients are not confined to the bed, nor even to their rooms, and perhaps throughout the entire course of the disease are able to transact a certain amount of business. Such cases have been called “walking cases” of typhoid fever.

The eruption appears in these cases early, is of short duration, only a few spots appear; usually there is only one crop. Diarrhœa is also present in most cases of this class, but it is of a mild type, the discharges from the bowels apparently giving relief to the patient. In some cases the diarrhœa alternates with constipation, or constipation may be present throughout the entire course of the disease, and the cases go on exhibiting a varying amount of fever for from twenty to thirty days, until gradually convalescence is established. This class of cases, if properly managed, rarely prove fatal; but, if improperly managed, there is great danger. For instance, if a patient walks about while he is suffering from one of these so-called mild attacks of typhoid fever, he does it at great risk to life—in other words, there should be no “walking cases” of typhoid fever. A patient sick with typhoid fever, however mild the type, should take to his bed and remain there until convalescence is fully established, as it is impossible to say just how extensive the changes may be that have

occurred in the intestinal track, and in the mildest type of the disease they may be of such a nature that very little physical exertion will cause intestinal perforation, which will be followed by a fatal peritonitis.

The *abortive form of typhoid fever* is ushered in with all the symptoms of a typical case—headache, lassitude, pain in the limbs, nausea, etc.—and the temperature during the first week follows the regular variations of the fever. At the onset the disease has every appearance of a severe form of typhoid fever; the temperature may rise as high as 105° F. or 106° F. by the end of the first week; delirium is often active, and diarrhœa is present. By the end of the second week, certainly by its close, if recovery occurs, the fever is cut short, and abruptly disappears; the temperature falls to the normal standard, and the patient passes on to a state of rapid and complete convalescence. The eruption, diarrhœa, and all the urgent symptoms of the disease may be present, and yet before the end of the second week the patient may have fully convalesced. That it is the typhoid poison which thus acts upon the system, and gives rise to the characteristic symptoms of typhoid fever in these abortive cases, is evidenced by the fact that at the post-mortem examinations the characteristic typhoid intestinal lesions are found, and these, taken in connection with the presence during life of the typhoid eruption, establish the diagnosis beyond question. There can be no doubt but that an individual may be affected, overwhelmed, as it were, by typhoid poison, and yet not develop well-marked typhoid fever. So, if only a moderate amount of typhoid poison is introduced into the system, a mild or an abortive type of fever will be developed. The natural powers of the individual to resist the action of such poisons must always be regarded, and should be taken into consideration in the treatment of a case.

DIFFERENTIAL DIAGNOSIS.—In a typical case, after the fever is fully developed, the diagnosis is not difficult. The presence of febrile excitement, marked by evening exacerbations and morning remissions, headache, diarrhœa, abdominal tenderness, and other abdominal symptoms, and the

presence of the characteristic rose-colored spots, are sufficient for a diagnosis.

In the mild type of the disease, or when the symptoms are developed irregularly, or during the first week of a typical case, the diagnosis is often difficult, and sometimes impossible. The principal diseases which are liable to be confounded with typhoid fever are typhus and relapsing fevers, typho-malarial fever, acute tuberculosis, pyæmia, septicæmia, pneumonia, and gastro-enteritis.

The points of differential diagnosis between typhoid and typhus, relapsing and typho-malarial fevers, will be more apparent, and more readily comprehended, after we have studied these different forms of fever. I shall therefore not call your attention to their differential diagnosis until I have given you a history of these fevers.

ACUTE TUBERCULOSIS.—This disease is attended by very many of the symptoms which are present in, and by some supposed to be characteristic of typhoid fever. The fever of acute tuberculosis is of a remittent type, attended by evening exacerbations and morning remissions, delirium, a dry, brown tongue, a tendency to stupor, great prostration, rapid emaciation, and sometimes by a diarrhœa, with abdominal tenderness and tympanitis. All of these are among the prominent symptoms of typhoid fever; consequently these two diseases are frequently mistaken the one for the other. More than once have patients in Bellevue Hospital, with the diagnosis of typhoid fever, presented at the post-mortem examination the characteristic lesions of acute tuberculosis. If, therefore, patients with acute tuberculosis may go through a large general hospital, under the observation of diagnosticians, who certainly are not men of inferior ability, and be supposed to have typhoid fever, there evidently is great danger of a mistake in diagnosis.

The higher range of temperature in acute tuberculosis than in typhoid fever is one of the distinguishing characteristics of the disease. Usually, early in the progress of the disease, it reaches 106° F. or 107° F., while in typhoid fever the temperature rarely reaches 106° F., and even then in

most cases not before the end of the second week of the fever, by which time you will have been able to determine the true nature of the disease. Again, you will notice that there is no eruption, neither is there enlargement of the spleen in acute tuberculosis, while both are very constant attendants of typhoid fever; yet their absence is not positive proof that typhoid fever does not exist.

In all doubtful cases you must take into account the family history of the patient, his immediate surroundings, whether typhoid fever is prevailing at the time, and whether the patient has been exposed to typhoid poison. These are important points, and by a careful study of them, if you are able to watch the case throughout its entire course, probably you will arrive at a correct diagnosis before the end is reached. Should you see the case during the first week of the disease, rely upon the presence of the rose-colored spots for a diagnosis of typhoid fever, and you will rarely mistake it for acute tuberculosis.

PYLEMIA AND SEPTICEMIA.—These diseases, while developing, present many symptoms which resemble those of the developing stage of typhoid fever. In most cases you will be able readily to recognize them, as the surface of the body has a jaundiced hue; there are no lenticular spots, and the febrile symptoms are irregular in their development. There are exacerbations and remissions, but their appearance and disappearance are not marked by any regularity, and usually there is more than one exacerbation and remission in the twenty-four hours. Not only are the variations in temperature irregular, but the temperature reaches a high degree much sooner, and ranges higher throughout its entire course in pyæmia and septicæmia than in typhoid fever. In pyæmia and septicæmia you will also have early in the disease profuse sweatings, great prostration, rapid emaciation, delirium, subsultus, tympanitis, and diarrhœa, while in typhoid fever these do not come on until late in the disease. Besides, the history which precedes and attends the development of pyæmia and septicæmia widely differs from that of typhoid fever.

There is a condition of septic poisoning occasionally met with, resulting from the introduction into the system of septic malaria through the drinking water, which so closely resembles that which is the result of typhoid poisoning that it is almost impossible to make a differential diagnosis. In these cases the absence of the rose-colored spots is almost the only distinguishing feature.

PNEUMONIA.—Pneumonia, with typhoid symptoms, is sometimes mistaken for typhoid fever. It is called in your books *typhoid pneumonia*. The differential diagnosis is not difficult if you remember that the pneumonia which complicates typhoid fever does not come on until late in the fever, and you have the regular history of typhoid fever preceding its development. On the other hand, when the typhoid symptoms are present from the beginning of, or come on at the end of the second stage of the pneumonia, the physical signs of the pneumonia will attend or precede the typhoid symptoms. There will be cough and the characteristic pneumonic expectoration; there will be no eruption, and no typical variation in temperature.

If you do not see a patient who is over sixty years of age with this type of pneumonia until the second or third week of its progress, although evidences of lung consolidation may be present frequently, it will be very difficult to decide whether the pneumonia is or is not complicating a typhoid fever, and under these circumstances of course the differential diagnosis will be very difficult.

GASTRO-ENTERITIS.—In the adult this disease is quite readily distinguished from typhoid fever, as the diarrhœa and vomiting precede the febrile movement; the fever is irregular in its development and progress, and the temperature rarely rises higher than 103° F. In a child between two and six years of age it is very difficult to distinguish gastro-enteritis, or intestinal catarrh, as it is sometimes called, from typhoid fever. The eruption is not so prominent or constant a symptom in the child as in the adult, and with both diseases we have diarrhœa, tympanitis, and typhoid symptoms. These circumstances render many cases of this character difficult of diagnosis. When all

the symptoms precede the fever, and you can have a history of the case, and a thermometrical record from the beginning of the fever, in most cases you can readily make the diagnosis; but if you do not see the case until it has reached the second week of its progress, and you have no accurate or reliable history of its development, a positive diagnosis is impossible.

TRICHINOUS DISEASE.—Poisoning by trichinæ has frequently been mistaken for typhoid fever. This condition is not unfrequently attended by diarrhœa, vomiting, and the development of other typhoid symptoms; but with poisoning by trichinæ there is almost constantly present muscular pains and œdema of the eyelids, which will be sufficient to arrest your attention. We have, then, in poisoning by trichinæ, diarrhœa, vomiting, tympanitis, rapid emaciation, great exhaustion, a brown, dry tongue, high temperature, and other typhoid symptoms; with these you have the œdema of the face, especially of the eyelids, and the most intense muscular pains. By removing a small portion of the muscular tissue and placing it under the microscope, the trichinæ can be seen, and thus you will be enabled to make a positive diagnosis.

LECTURE V.

TYPHOID FEVER.

Prognosis.—Duration.—Relapses.

I HAVE already spoken to you of the differential diagnosis of typhoid fever, and will now give you some of the more prominent rules which should govern you in its prognosis.

PROGNOSIS.—Death may occur at any stage of this fever. A typhoid patient is not out of danger until all tympanitis, diarrhœa, and other abdominal symptoms which indicate that intestinal changes are still progressing, have disappeared. Independent of complications the duration, type, and intensity of the febrile excitement has more to do than all the other elements in determining the prognosis in any case of typhoid fever. The height of the temperature on the eighth day determines the range of temperature that may be expected on each succeeding day. If upon that day it is not higher than 104° F. or 105° F., and has been regular in its development (independent of complications), the prognosis is good; in uncomplicated cases it very rarely rises higher than the degree it has reached at that time. A prolonged high temperature (above 105° F.) after the first week renders the prognosis unfavorable.

In mild cases, during the second week, a marked morning remission occurs, which begins early and continues until midday; the evening exacerbation is late, and by the end of the second week there is a marked and permanent fall in the temperature. In severe cases, the opposite conditions are observed. A sudden rise in temperature, or a rapid and extreme fall at any period of the fever, is a very bad omen;

the latter often precedes the occurrence of a severe intestinal hemorrhage. Marked variation from the typical temperature of the disease indicates the existence of complications. Slight decline, accompanied by great fluctuation of temperature, during the third week, is an unfavorable symptom. The natural power of an individual to resist disease, especially the effects of prolonged high temperature, is a very important element in prognosis. The organ which is the surest indicator of such power (especially in typhoid fever) is the heart. If the pulse is full and regular, perhaps beating at the rate of 110 or 115 per minute, if the cardiac impulse is good, and a distinct first sound can be heard, even though at the end of the second week the temperature stands as high as 106° F., the prognosis is favorable. If, however, the pulse has risen to 120 or 130 per minute, if the apex-beat is feeble or imperceptible, and the first sound of the heart is indistinct or altogether obscured, with a tendency to cyanosis and pulmonary œdema, the indications are that the patient's powers of resistance are failing, and under such circumstances the prognosis must be unfavorable. It is not so much the rapidity as the regularity, *a sudden falling and a sudden rising of the pulse, that indicates the impending danger.* The rapid rising of the pulse upon the slightest excitement is the most unfavorable indication, as it shows extensive heart-failure and a rapid giving way of vital power.

A sudden fall of the pulse from any cause must always be regarded as an unfavorable indication. The abundance or color of the eruption does not influence the prognosis. Excessive tympanitis and severe abdominal pains are unfavorable symptoms.

Severe and protracted muscular tremors, with subsultus, indicate danger. Sudden collapse during the second and third weeks of the fever is always attended with danger, as it is very likely to be due to copious intestinal hemorrhages or intestinal perforation. It sometimes occurs independently of either of these causes, but nevertheless is very apt to be soon followed by a fatal result.

The prognosis is always bad in persons who are very fat,

and in those who are the subjects of gout, diseases of the kidney, or any other severe form of chronic disease. In all such persons, during the second and third weeks of the disease, you must constantly be on the watch for the occurrence of sudden collapse.

Different opinions have been given as to the importance of intestinal hemorrhage in reference to prognosis. Some have regarded slight intestinal hemorrhages as beneficial, while others have regarded them as always of dangerous significance.

My own experience leads me to the belief that when the hemorrhage is scanty it has little influence on the final result. When it occurs before the twelfth day of the fever, it often does good by relieving the intestinal congestion. But when profuse, or even a slight hemorrhage after the twelfth day, is an unfavorable symptom and renders the prognosis unfavorable. The occurrence of the hemorrhage renders it probable that ulceration has extended to the vessels beneath the transverse muscular fibres of the intestine, and such ulceration is very apt to go on to perforation and a fatal peritonitis. So that although the patient may survive the hemorrhage, there is great danger of death from peritonitis, and this danger must always enter into your prognosis, whether the hemorrhage is slight or severe.

The influence of age is very great in determining the prognosis in any case of typhoid fever.

The prognosis is much better in children than in adults. Occurring in persons over forty years of age, the prognosis is decidedly unfavorable, even though the symptoms may not indicate a severe type of the disease.

In the case of those individuals who habitually use alcoholic stimulants, whose power of resistance to high temperature is diminished, the rate of mortality is very great.

The puerperal state renders your prognosis especially unfavorable. The danger to the patient is equally great, whether the fever comes on prior to delivery or during puerperal convalescence.

In this fever there is greater danger to those who are suf-

fering from any form of chronic disease than to those who are in a healthy condition at the time of the attack.

Without delaying you longer with those conditions in the ordinary course of the disease which influence its prognosis—the most important of which have been referred to under the head of symptoms—I will pass to the consideration of the complications which influence its prognosis. They are more numerous than those in any other disease.

I will first briefly allude to those which are intimately connected with, or dependent upon, the morbid changes ordinarily incident to the disease, and afterwards speak of those which may be designated as accidental complications.

The parenchymatous changes which take place in the different organs of the body, during the progress of this fever, necessarily influence prognosis. For instance, the muscular degenerations of the cardiac walls and the consequent loss of heart-power, which favors pulmonary and other hypostatic congestions, and the diminished quantity of blood sent to the various tissues of the body, interfere more or less with their nutrition. Necrotic and gangrenous processes, sometimes met with in the cellular tissues of the surface and along the line of the intestines, also the venous thrombi which so frequently develop in a protracted case of this fever, are, to a certain extent, the result of this cardiac weakness. It is apparent that the development of extensive cardiac degenerations must render the prognosis unfavorable.

Excessive cardiac weakness favors the development of blood-clots in the heart-cavities; these may break up and cause embolism somewhere in the course of the general circulation, and thus lead to changes which may destroy life. Again, *intestinal perforations*, one of the results of the intestinal changes incident to the fever, render the prognosis most unfavorable. The same is true of copious *intestinal hemorrhages* coming on after the third week of the fever, as well as of all those glandular changes which are a part of the natural history of the fever, and which I have already described.

Any of these changes may lead to complications which

endanger the life of the patient, and consequently, when they occur, necessitate a guarded, if not an unfavorable prognosis.

Some of the prominent accidental complications which may occur in the course of typhoid fever, but which do not belong to its regular history, have their seat in the respiratory organs. Slight bronchial catarrh is present in nearly every case, and can hardly be regarded as a complication. It is so much a part of the clinical history of the disease, that some have named this fever *bronchial typhus*. There is another much more serious bronchial complication, namely, catarrh of the smaller bronchi, or capillary bronchitis. This usually comes on during the second or third week of the disease, and if extensive, greatly endangers the life of the patient. If, then, during this period of the fever, you have subcrepitant râles suddenly developed over the whole of both lungs, accompanied by great dyspnœa and an abundant expectoration of stringy mucus, you are warranted in giving an unfavorable prognosis.

Extensive œdema of the lungs occurring with, or independent of, capillary bronchitis and pulmonary congestion, sometimes comes on suddenly during the third week of typhoid fever, and indicates great failure of heart-power. The slightest indication of its occurrence should always be regarded with suspicion. It is not unfrequently accompanied by more or less extensive hemorrhagic infarctions of the lungs; these depend on embolism of some of the branches of the pulmonary artery due to fragments of clots which have formed in the right side of the heart, the result of the cardiac weakness; these often lead to gangrene of the lung. It is sometimes impossible to diagnosticate their existence during life.

Pneumonia, when it complicates typhoid fever, is generally latent. It comes on very insidiously, and unless you are on the watch for its development, and make frequent and careful physical examination, it will pass unrecognized. It is more frequently developed during the third and fourth week of the fever, and usually is catarrhal rather than croupous in character. At first only single lobules are in-

volved, but after a time an entire lobe becomes consolidated. When irregular variations in temperature occur during convalescence, or during the third or fourth week of the fever, there is reason to suspect the development of pneumonia. In the majority of cases the characteristic pneumonic cough and expectoration are absent. Whenever an extensive pneumonia complicates typhoid fever, the prognosis is especially unfavorable.

Pleurisy does not occur so frequently, as a complication of typhoid fever, as does pneumonia or bronchitis. When it does occur, the almost invariable product of the inflammatory process is pus. Usually it comes on late in the disease, comes on insidiously, and is quite likely to pass unrecognized unless frequent physical examinations of the chest are made. In many instances it is really a sequela of the fever, not developing until three or four weeks after the fever has run its course. Its occurrence must always be regarded as unfavorable, for a year or even longer time must elapse before recovery can take place, and even then recovery is doubtful.

Occasionally laryngitis is a serious complication of this fever. It generally occurs in those cases where the fever has been very protracted, and there is great prostration. Its presence is marked by sudden and very intense inflammation of the mucous membrane of the glottis, which is liable to become œdematous, when death may suddenly occur. It may lead to ulceration of the mucous membrane. Whenever, during any stage of a typhoid fever, the characteristic symptoms of laryngeal obstruction occur, remember the danger of œdema glottidis and of extensive laryngeal ulceration, and promptly resort to those means which shall relieve the unpleasant symptoms, and avert the danger which threatens your patient.

Pyæmia may be met with as a complication during convalescence from typhoid fever, but it is not of as frequent occurrence as septicæmia. Whenever we have septic poisoning developed, with extensive sloughs in the intestines, the prognosis is exceedingly unfavorable.

Acute gastric catarrh is another complication of this fever,

the possible occurrence of which must enter into your prognosis. A patient may have reached his fourth week, and be rapidly convalescing, his desire for food returning ; you endeavor to hasten his recovery by increasing the quantity of food taken, or by allowing him to partake freely of such articles of food as are difficult of digestion. The result of this overcrowding, or of imprudence in diet, is irritation and inflammation of the enfeebled gastric mucous membrane. Vomiting of a stringy mucus occurs, which by its prostrating effects endangers or destroys the life of your already enfeebled patient. I would impress you with the importance of exercising the greatest care in regard to the diet of patients convalescing from typhoid fever. They should be restricted to milk and nutritious broths in moderate quantity until all danger from this complication shall have passed.

Disturbances of nerve-function have been considered under the head of symptoms, but, not unfrequently, certain brain and nerve lesions are developed which cannot be classed under that head.

Cerebral œdema may complicate a typhoid fever during its third week, and give rise to symptoms of a grave character. A decided enfeebling of the mental powers and a tendency to stupor announces its occurrence.

Hemorrhagic extravasations on the surface and into the substance of the brain, the result of degeneration of the walls of the cerebral vessels, occasionally occurs during the height of the fever. If the effusion is moderate, no marked symptoms are developed ; but if a considerable extravasation takes place, it gives rise to symptoms of cerebral compression.

Meningeal inflammation is a rare complication.

The occurrence of any of these complications in any case renders the prognosis unfavorable.

You must remember that during the second or third week of the fever certain cerebral disturbances may occur, which seem to indicate the existence of some one of these complications, when really no cerebral lesion exists. Usually, these are present in patients who have had a continuously

high temperature ; in favorable cases they disappear after a few days. These have been referred to under the head of symptoms.

You will encounter various other disturbances of the nervous system, such as hemiplegia, paraplegia, etc., which may simulate those due to lesions of nerve-centres, or local forms of paralysis and anæsthesia, which seem to be confined to individual nerves ; but as these functional disturbances do not depend upon any anatomical changes, the prognosis in such cases is good.

Those changes in the kidney due to the parenchymatous degeneration which usually attends this fever, have been already noticed ; but occasionally nephritis is developed as a sequela. The urine becomes scanty, is loaded with albumen, and contains blood and casts ; the face and extremities become œdematous, and death may occur from uræmia. The occurrence of this complication necessarily renders the prognosis bad.

In a few instances under my observation, severe catarrh of the bladder has developed during convalescence, greatly complicating the case ; in one instance the cystitis was accompanied by pyelitis.

Suppurative inflammation of the cellular tissue of the body, or cellulitis, especially of the surface, often complicates convalescence, and in some cases causes death. It is most liable to develop in those parts which have been subjected to long-continued pressure. Occasionally it is met with in the pharynx and along the line of the lymphatics.

Accompanying these cellular inflammations, or occurring independently of them, not unfrequently gangrenous inflammations of the integument occur, giving rise to what has been called *bed-sores*. These gangrenous processes are most frequently developed at those points which have been subjected to the greatest pressure, on account of the position of the patient in bed, such as the sacrum, nates, heels, and shoulder-blades, etc. In the simplest form of *bed-sores* there is only a superficial loss of substance ; in more severe cases the subcutaneous cellular tissue is involved ; and in the worst cases the muscles and fibrous tissues. I have met

with cases where the slough had involved the connective tissue and muscles, and laid bare the bony tissue.

A considerable number of typhoid patients who have lived through the fever, die either from the exhausting effects of these bed-sores, or from the septic poisoning resulting therefrom.

The possible occurrence of these complications must enter into the prognosis in every severe case, and the earlier they make their appearance the greater the danger.

We have now completed the list of principal complications which are to modify your prognosis in any case of typhoid fever. Before leaving the subject, I will say a word in regard to the *duration* and *mode* of termination of this fever.

DURATION.—Its average duration is from three to four weeks; it may terminate in death or recovery at an earlier date. A typical case extends over a period of four weeks. The period of invasion lasts from one to five days. The period of glandular enlargement continues until about the fourteenth day. The period of ulceration extends from the twelfth or fourteenth day to sometimes between the twenty-first and twenty-eighth. When the fever is protracted beyond the middle of the fourth week, in most instances this is due to some complication, or to an extension of the intestinal ulceration. The period of greatest danger is at the close of the third week. Death rarely occurs before the fourteenth day. The prominent direct causes of death are: *First, Toxæmia; Second, Asthenia; Third, Suppression of the excretory function of the kidneys; Fourth, Hyperæmia and œdema of the lungs; Fifth, Intestinal hemorrhage; Sixth, Exhaustive diarrhœa; Seventh, Intestinal perforation; Eighth, Peritonitis, with or without intestinal perforation.* In nearly all cases the failure of heart-power is directly or indirectly the cause of death. In no case can convalescence be said to be fairly established until the temperature remains normal for two successive evenings. Its termination, like its commencement, is gradual, and it is not marked by any critical evacuation or day of crisis.

RELAPSES.—After typhoid fever has run its course, and

after the patient is entirely free from fever, quite frequently we have a new development of the fever; these new developments are called relapses. Their course corresponds with that of the primary attack, only they are of shorter duration. The temperature rises more rapidly, the eruption reappears, the spleen enlarges, the intestinal and abdominal symptoms return, and all the prominent symptoms of the primary fever are rapidly developed. As a rule, the relapse is milder than the primary attack. If it terminates fatally, the post-mortem examination shows, in addition to the cicatrizing intestinal ulcers of the primary attack, the recent intestinal changes of the relapse. The lesions of the relapse, although of the same character as those of the primary attack, are less extensive.

It is very difficult to give a satisfactory explanation of these relapses. Some claim that they are the result of certain plans of treatment, especially the cold-water plan. This assertion lacks proof. Again, others hold that all relapses depend upon a new infection. Perhaps this is possible if the patient remain in the same locality and has the same surroundings as when he had the primary attack; but how shall we explain relapses in those who are removed from all the sources of the primary infection? Another explanation offered is that a part of the typhoid poison has remained in the system, undeveloped during the primary attack, and that some time after this has passed the poison reproduces itself and sets up a second fever.

A more recent theory is, that the typhoid poison thrown off in the faces of the patient is reabsorbed and causes the relapse. Unquestionably, it is possible for healthy glands to become inoculated by sloughs thrown off from those first affected.

In many cases it is impossible to account for the occurrence of the relapse, and all of these explanations as to the cause in any case are more or less unsatisfactory.

In those cases which have come under my own observation, I have noticed that the splenic enlargement which has existed during the course of the fever does not subside with its decline; and that the tenderness along the line of the

intestines, especially in the right iliac region, continues during the period between the original attack and the relapse. In some instances, apparently, the relapse has been brought on by indiscretion in diet, or by injudicious exercise on the part of the convalescent patient. Occasionally relapses have occurred when great care had been taken against any indiscretion or over-exertion.

There is little doubt but that relapses are of much more frequent occurrence in those cases that are treated with cathartics during the first week of the fever, than in those where cathartics are not employed.

LECTURE VI.

TYPHOID FEVER.

Treatment.

BEFORE speaking in detail of the treatment of typhoid fever, I will say a few words concerning its prevention.

If the modern theory (which I have already given you) of its etiology be accepted, the question naturally arises, cannot the typhoid poison be prevented from entering our dwellings, or polluting our drinking-water?

Facts prove almost conclusively that typhoid fever is never of spontaneous origin. Should it occur in the locality where you may reside, if possible find out its origin. If no case has ever before occurred in the locality, endeavor to ascertain the manner in which the typhoid poison has been introduced. If it is already endemic, limit the disease to the first few cases by a most thorough disinfection, and remove all those surroundings which favor the reproduction of the typhoid poison.

If the theory is correct, that typhoid fever is dependent upon a poison contained in the excrements of a typhoid patient, then the poison should be destroyed as soon as it is discharged from the body. For this purpose, the intestinal discharges should be received into a porcelain bed-pan, the bottom of which should be covered with a thin layer of powdered sulphate of iron; immediately after the discharge, crude muriatic acid, equal in quantity to one-third of the faecal mass, should be poured over it. Never empty the discharges of a typhoid patient (no matter how thoroughly

they may have been disinfected) into the privy or water-closet used by the family. Trenches should be dug for their reception, and new trenches should be opened every few days; the greatest care should be taken that these trenches are not so situated that drainage from them can contaminate wells or springs which furnish drinking-water. All under-clothing or bed-clothing that may have become soiled by the discharges from the bowels, should be immediately immersed in chlorine water, and thoroughly boiled within twenty-four hours. This procedure will certainly destroy the infective power of the typhoid poison contained in the intestinal discharges, and in the majority of instances you will prevent the spread of the fever.

Repeated observation shows that when one member of a family has typhoid fever, not unfrequently it is developed in every other member. This spread of the disease can be prevented, unless there is some local cause for its development which cannot be reached.

When its origin is not apparent, the wells, springs, and all the sources from whence water is derived for drinking and cooking purposes should be carefully and thoroughly inspected. Care must be taken that the waste-pipes from wells and springs do not pass directly into cesspools or sewers, and thus become a means for the conveyance of impure gases into the springs and wells.

The greatest care must also be exercised in regard to home drains and sewer-pipes, that they shall be free from leakage and obstruction, and that all water-closets, sinks, and other openings into them be provided with suitable traps.

When unpleasant odors are constantly present in dwellings, especially in sleeping apartments, disinfectants should be employed, and the house be thoroughly ventilated.

When it may be necessary to open drains and cesspools in a dwelling for purposes of repair or cleansing, the same precautions should be exercised; these are especially of importance during the summer and autumn.

In conclusion, let me impress upon you this fact, that when typhoid fever is carried from the sick to the healthy, the evacuations are the chief, if not the only means of con-

tamination; consequently, the importance of thoroughly disinfecting the excrements of typhoid patients should always be borne in mind.

In this connection the question naturally arises, can we not counteract or neutralize the effects of the fever poison after it has gained admission into the system, and thus prevent the development of typhoid fever? To accomplish this, at one time blood-letting was resorted to; but at the present day few practitioners would venture to suggest such a plan of treatment, and few patients could be found willing to submit to it. Emetics were given on the supposition that the fever-poison acted primarily upon the mucous membrane of the stomach, and that the offending agent might be removed by their early administration, and thus its absorption into the system prevented. As it has been proved that the typhoid poison can be introduced into the system through other channels than the stomach, and as experience has shown that emetics have not the power to prevent the development of typhoid fever, their use has been abandoned. Diaphoretics have also been employed; but there is not the slightest proof that typhoid or any fever-poison was ever removed from the system by sweating. A patient with some of the premonitory symptoms of fever may sweat, be relieved, and at once recover, but such a patient has not received the typhoid poison into his system, and was not, as is sometimes said, "threatened with typhoid fever."

Notwithstanding the bold affirmation of the author of the cold affusion plan of treatment, that if it were resorted to before the third day of the disease, it would invariably arrest its development, it has failed to stand the test of practical experience.

More recently, sulphate of quinine, administered in large doses, has been thought to have the power of arresting the development of typhoid fever in the same way that it arrests malarial fever, by its anti-periodic power; but there is no evidence that it has any such power, and as a prophylactic remedy it has been abandoned.

I might go on almost indefinitely enumerating measures

which have been resorted to for preventing the development of this fever; but after the poison has once gained entrance into the system, no means have as yet been discovered by which it can be counteracted or neutralized so as to prevent the development of the disease. The duty of the physician is to guide the disease, so far as he may be able, to a favorable issue, and prevent injury to organs essential to life, keeping in mind that a certain definite period must elapse before this result can be accomplished.

Before entering into a detailed account of the treatment to be pursued in the management of a case of typhoid fever, I will say a few words in reference to the *arrangement of the sick-room* of fever patients. Though often overlooked, this is a matter of no inconsiderable importance, not only as regards the comfort of the patient, but it has much to do with the successful issue of the case.

It is of the greatest importance that a properly qualified nurse be selected; one who has had experience in the care of fever patients is to be preferred. In the next place, the patient should be placed in a large and well-ventilated apartment. All furniture should be removed from the sick-room, except those articles which are necessary for the comfort of the patient and the convenience of the attendants. Remove the carpets from the floor, place your patient in a bed of moderate size in the centre of the room, and let there be free ventilation during both day and night.

The temperature of the apartment (if possible) should be kept below 60° F.

The bed and body linen of the patient should be changed daily, and at once be removed from the sick-room and placed in a weak solution of chloride of sodium; especially is this important if the patient is having frequent discharges from the bowels. The apartment should be kept perfectly quiet, the light subdued, and only the attendants should be allowed in the room.

These preliminary arrangements having been made, we will suppose we have in charge a patient with a mild type of typhoid fever. All medicinal interference in such a case is unnecessary. The treatment resolves itself into the

arrangement of the sick-room and proper diet : milk is preferable, *fruits are not to be allowed in any case*. In the mildest case this care in diet is important, and the patient should be kept in bed until convalescence is fully established. This should be insisted upon in the mild as well as the severe cases.

As I have already stated, the temperature in a mild type of this fever rarely rises above 103° F. ; therefore there is no necessity for resorting to antipyretic measures ; frequent sponging of the surface with cold or tepid water, as is most agreeable to the patient, will be found of service.

By far the larger number of cases of this fever are of a more severe type, and though in your treatment you must be guided by the circumstances which attend each individual case, usually you will be obliged to resort to more decided measures.

Remember that there are no specifics for this disease ; all of those which have been proposed and employed have either fallen into disuse, or are resorted to only as aids in general treatment.

Typhoid fever is a disease that has certain stages to pass through, limited only by days and weeks. There is great doubt whether the physician can shorten its duration by a single day, but experience warrants the belief that many lives may be saved by remedial measures used at the proper time, and combined with judicious hygienic management.

There are critical periods in this disease : be prepared by knowledge and judgment to carry your patient (if possible) safely through them. Unquestionably one of the most important things to be accomplished is the reduction of temperature, or rather the keeping of the temperature below a certain standard. Blood-letting, emetics, diaphoretics, cathartics, chlorine water, and mineral acids have all been resorted to in order to reduce temperature. The last two agents were supposed to reduce temperature by neutralizing the typhoid poison. At the present day I think there is no intelligent physician who imagines he can neutralize the typhoid poison, and thus reduce temperature, while only a few years ago these agents were supposed to possess such

power, and were very extensively employed for such a purpose by some intelligent physicians.

The agents which more recently have been employed for this purpose, namely, sulphate of quinine and cold applications to the surface, are powerful agents in reducing the temperature and lessening the severity of the disease; but they can never shorten its duration, and if you employ them, expecting this result, you will be greatly disappointed. It is claimed by many very distinguished observers of the present day that the parenchymatous degenerations of the different organs and tissues of the body, which are found in those who die of typhoid fever, are due to the prolonged high temperature which is present during the course of this disease; but as yet there are no facts to prove this assertion, for the same parenchymatous changes are found in the bodies of those who have died of diseases, the course of which was not marked by high temperature, and did not extend over a period of more than forty-eight hours. So far as we are able, to determine by analogy upon what these parenchymatous changes depend, we are led to believe that the specific poison of the disease has more to do with their development than the high rate of temperature. One thing must be apparent to every clinical observer: that the injurious effects of a prolonged high temperature are early and most markedly shown by disturbances of the cerebro-spinal system. It is still an unsettled question whether these disturbances are due to the primary changes in the constituents of the blood, which always accompany a high range of temperature, or to the direct effects of the high temperature on the nerve centres.

Whichever view we accept or adopt, the employment of those means which have the power of safely reducing temperature is indicated, and when judiciously used they have much to do with the safety of the patient.

All those means which have been employed for the reduction of temperature are included under the general term of *antipyretics*, and the treatment of disease by the use of these agents has received the name of *antipyretic treatment*.

Unquestionably the most efficient and reliable of the antipyretic agents are the external application of cold by means of baths, packs, and effusions, and the internal administration of the sulphate of quinine. The quinine is not administered to produce any specific action upon the typhoid fever poison, but is employed for its antipyretic power. There are other antipyretic agents besides these two, but they are of so little importance that it is necessary to give them only a passing notice after we shall have considered these two important ones.

At the present time the opinion prevails, to a great extent, that the application of cold to the surface is the great antipyretic in the treatment of fever. This is no new teaching. Long ago Dr. Currie recommended the application of cold to the surface of the body for the purpose of rapidly reducing temperature, and proved that it had such an effect: yet it was never very generally practised, and soon fell into disuse, as there was nothing reliable to guide one in its application. As we now have the thermometer to guide us in its application, more recently it has been resorted to with considerable success.

I will give you some general rules, which may be of service to you in the use of this antipyretic in the treatment of typhoid fever.

As soon as the axillary temperature in the evening rises above 103° F., place the patient in a water-bath having a temperature of 70° F. or 80° F., and gradually lower that temperature by the addition of cold water or ice, until the temperature of the patient begins to fall. You may be compelled to lower the temperature of the bath to 60° F. before the temperature of the patient is affected: but the lowering of the body temperature must be accomplished by the lowering of the temperature of the bath, taking care that the latter does not fall below 60° F. When the temperature begins to fall, renew your thermometrical observations every two or three minutes. While the baths are being used, the temperature must be taken by placing the thermometer in the rectum. If it falls rapidly—that is, two or three degrees in five or six minutes—as soon as the

fall has reached 103° F. remove your patient from the bath ; if it falls slowly, as soon as it reaches 101° F. he should be removed and immediately placed in bed. Never keep the patient in the bath until the temperature shall have reached the normal standard ; should you do so, he may pass from a condition of fever into a state of collapse, as the temperature continues to fall for some time after his removal from the bath. While in the bath, cold should be applied to the head by means of a sponge wet in cold water or by an ice-bag.

The cold pack is much less effective than the bath ; but if the patient is too feeble to be moved, it may be employed with benefit. You should wrap the patient in a sheet wrung out of tepid water, and over this sheet apply one wrung out of cold water. The latter may be removed as often as it becomes warmed ; its application and removal may be continued until the desired fall in temperature shall be obtained.

In severe cases, during the first and second weeks, you will find that after the temperature has been reduced by the application of cold to the surface, it will begin slowly to rise until it reaches its former height. Usually one to three hours will elapse before it begins to rise, and from two to six before it reaches its former height. You will then be obliged to repeat the baths or packs, and to continue their use, both day and night, from three to six times during the twenty-four hours, if you expect to keep the temperature below 103° F., and accomplish anything by this plan of treatment. My experience in the use of cold applications leads me to believe that unless you are able to maintain a low range of temperature after four or five baths, you gain very little by their continuance. In other words, if, after using the baths for twenty-four hours, the temperature of your patient rapidly rises to the same or a higher degree than it was before their use was commenced, you will obtain little or no benefit from their continuance unless you can introduce some other agent which shall maintain the low temperature reached by the bath. I am also convinced that, after the second week of typhoid fever, cold baths should not be employed to reduce temperature, for by their continuons use after that period they may do

great harm. The condition of a typhoid patient during the first and second week of the fever is very different from that during the third and fourth week. During this latter period there is great danger of collapse after a cold bath, and in several instances I am confident that pulmonary complications have been the result. In a few instances the temperature can be very rapidly lowered by the application of ice-bags to the abdomen. The rapidity with which the temperature can be reduced usually depends upon the severity of the fever. In some cases, when the patient is placed in the cold bath, the temperature will immediately begin to fall; in other cases there will be a gradual reduction of temperature as the water is made cooler. In certain severe cases, you may keep a patient in a bath of the temperature of 60° F. for the space of half an hour without the temperature falling a degree. These cases are exceedingly grave in character, and you should use the bath with great care.

Finally, let me impress upon you that in typhoid fever, in order to reduce the temperature, you must not indiscriminately apply cold to the surface of the body. Perhaps there is *no remedial agent which requires greater care and judgment in its use*; yet doubtless, when judiciously employed, the lives of many typhoid patients may be saved, and it is equally certain that when injudiciously employed, many lives may be destroyed. If you use the cold baths in conjunction with other means for reducing temperature (concerning which I will speak at my next lecture), I am confident you will accomplish much; but if you rely only upon the baths, in the majority of instances you will be disappointed in the result. At the present time it seems to me, that by some the benefit and power of cold baths in the treatment of typhoid fever have been overrated.

The general condition of your patient and the stage of the fever must be considered; also the effects of the first few baths must be carefully noted.

Should a patient's temperature range at 104° F. or 105° F., there is no positive evidence that you must resort to a cold bath, or that a cold bath is the best agent to be em-

ployed for its reduction. Again, if the patient after the second or third bath is more quiet, has less delirium (if delirium previously existed), if his breathing becomes easy and natural, if the heart's action is more regular and forcible, and he falls asleep and perspires, there can be no question in regard to the beneficial effects of the bath. If, on the other hand, the bath is followed by feebler heart's action, by dusky cheeks, by rapid respiration, and by coldness of the extremities, from which condition the patient rallies slowly and imperfectly, you may be certain that, however high the temperature may range, you will do harm by continuing the baths. When the extremities are cold, or there is profuse hemorrhage from the bowels, or when, from any cause, there is great feebleness of the heart's action, and especially in the case of aged persons, cold baths are contraindicated.

Cold compresses or ice-bags applied to the abdomen, in addition to their beneficial effect on the intestinal changes which constitute such an important element in the history of this fever, often have great power in reducing the general heat of the body. I have also in some instances found the body temperature rapidly lowered by injections of ice-water into the rectum. Care must be exercised that the cold injections are not administered too rapidly or in too large quantities.

Although this mode of abstracting heat and the lowering of the body temperature is never so effective as by baths and packs, still it has this advantage, that no such compensating increase in the production of heat follows the use of the cold injections as follows the cooling of the external surface by the baths.

In many cases the extreme obstinacy of the fever, which resists the most systematic use of cold, as well as the fact that some patients cannot bear a sufficiently frequent repetition of them to effect the desired result, or that there may be contra-indications to their use, necessitates the employment of other means for the reduction of the body temperature. To these I shall invite your attention at my next lecture.

LECTURE VII.

TYPHOID FEVER.

Treatment (continued).

WE have already considered the antipyretic power of cold applications in the treatment of typhoid fever, and I will now call your attention to the antipyretic power of the sulphate of quinine.

When quinine is employed as an antipyretic, it must be given in large doses; the administration of two grains every two hours, or a larger quantity administered in divided doses within a period of twenty-four hours, will not act as an antipyretic; but thirty or forty grains must be administered within a period of two hours.

If the stomach is irritable, and you fear that a large dose will produce vomiting, ten grains may be given every half hour until the desired quantity has been administered.

Usually from four to six hours after the antipyretic dose has been taken, the fall in temperature will begin, and in about twelve hours it will reach its minimum height; then it will remain stationary from twelve to twenty-four hours. After the temperature has once been reduced by the quinine, its administration may be discontinued until the temperature shall again rise to 105° F. As a rule, the temperature rarely ranges as high as before the quinine was administered.

This mode of administering quinine in antipyretic doses to fever patients rarely produces any symptom of cinchonism, other than a transient deafness after the first dose. In a large number of cases the temperature can be kept

below 103° F. by the sulphate of quinine; but in very severe cases it will be advisable, and sometimes it will be absolutely necessary, to employ not only the quinine, but at the same time the cold baths. My rule is, after I have reduced the temperature to 101° F., or 102° F., by a cold bath, to administer an antipyretic dose of quinine, and thus delay the recurring rise of temperature. While the cold bath more rapidly reduces temperature, the effect of the quinine is more lasting; consequently, by making use of both of these reliable antipyretics during the first two weeks, you will be able to control the temperature during that time. After this period it is not safe to resort to cold baths; but when the temperature rises above 103° F., occasionally you may use the cold pack in connection with antipyretic doses of quinine. If, during the third and fourth weeks, you fail to reduce the temperature by these means, administer during the twenty-four hours from ten to twenty grains of powdered digitalis—unless the pulse is very frequent and irregular—when its use is contra-indicated. As an antipyretic, digitalis should be administered only when quinine is given. It seems to increase the antipyretic power of the quinine, but has little or no power when administered alone.

The use of all these antipyretic remedies must be persisted in until the desired end—the reduction of temperature—is accomplished; but the peculiarities of each patient must be studied, and these agents must be so administered as to suit each individual case.

You cannot trust to the judgment of nurses and attendants, but you must determine for yourself what are the requirements in each case.

The satisfactory results obtained by the systematic use of these remedies justifies their employment; but the exact rules which are to govern one in their use, as to manner and time, can only be determined by experience.

All careful observers are aware that great danger attends prolonged high temperature; but it is still an unsettled question whether this danger is due to parenchymatous changes in the different organs, which some claim are the

result of the high temperature, or to disturbance of the nerve centres from the same cause. Whatever may be the final settlement of the question, the beneficial results which follow the antipyretic treatment of fevers are generally admitted; and my advice to each one of you is, at the outset of your professional career to make yourself perfectly familiar with the use of these most important and reliable antipyretics.

If you can keep the temperature of your patient at about 103° F. during the first two weeks of the fever, you have accomplished the *first* and perhaps the most important thing in the treatment of this disease.

Towards the end of the second, or during the third week, sometimes earlier, sometimes later, signs of failure of heart power begin to manifest themselves; the pulse becomes feeble and irregular; at times the surface is cool and moist; the patient complains of a sense of exhaustion, perhaps is unable to turn in bed; the tongue assumes a dry, brown appearance, and the necessity of supporting the patient becomes apparent. This will bring you to the *second* important question in the treatment of this fever, namely, *what means shall be employed to sustain heart power*, or, as is sometimes said, the vital powers of the patient?

When a patient, during the second or third week of the disease, dies from capillary bronchitis, pulmonary œdema, or suddenly passes into a state of coma, failure of heart power is the real cause of death.

In those cases in which, during the early part of the fever, you have been compelled to resort to a vigorous antipyretic treatment, during the third week, although the temperature may not rise higher than 101° F., the pulse frequently becomes extremely feeble, and reaches 140 per minute, the first sound of the heart becomes inaudible, muscular tremors, dry tongue, and all the phenomena which indicate failure of vital power are present. Under such circumstances the use of stimulants seems to be urgently demanded.

There are a few simple rules which may guide you in the administration of stimulants in this fever.

First.—They should never be administered indiscriminately—that is, never give a patient stimulants simply because he has typhoid fever.

Second.—When there is reasonable doubt as to the propriety of giving or withholding stimulants, it is safer to withhold them, at least until the signs which indicate their use become more marked.

Third.—In every case, but especially when stimulants are not clearly indicated, watch carefully the effect of the first few doses. There are few whose experience in the treatment of typhoid fever is such as to enable them to positively determine, from the appearance of the patient, when the administration of stimulants should be commenced.

Should you commence the administration of stimulants, it is necessary to see your patient every two hours, and note carefully the effect produced. If you find the tongue becoming dry, the patient more restless, the delirium more active, the temperature ranging higher, and the pulse more and more rapid, you may be certain that stimulants are contra-indicated. If, on the other hand, the pulse becomes fuller and more regular, if the first sound of the heart is more distinctly heard, or, if it has been absent, it has returned, if the restlessness and delirium are less marked, the tongue more moist and the patient more intelligent, you may be certain that the time for the administration of stimulants has arrived. When you have commenced their use, it is of the greatest importance that you administer them at stated intervals, especially during the night.

In a severe case of typhoid fever, a free administration of stimulants, just at a critical period (which may not last more than twenty-four hours), will often be followed by a refreshing sleep, and your patient may rapidly pass from an apparently hopeless condition to one of convalescence.

The *third* important thing to be accomplished in the management of typhoid fever patients is the maintenance of nutrition. You must bear in mind that the primary and principal effects of the typhoid poison are manifested in the changes which take place in the lymphatics of the gastro-

intestinal tract. Experience has taught us that the enfeeblement of the digestive and assimilative powers, due to these glandular changes, which are manifest from the very commencement of the fever, renders the digestion of solid food impossible, and for a long time it has been the rule of the profession to allow typhoid fever patients only liquid food.

There has been, and still is, great diversity of opinion in regard to the special articles of diet best suited to this class of patients. Most medical writers and practitioners claim that beef-tea is the proper diet for fever patients; consequently it is the rule to pour into these enfeebled stomachs a decoction of beef in such quantities as a healthy stomach could hardly tolerate, and which, in itself, has little or no nutritive element.

Others claim that gruels are far superior to animal broths, and advocate the feeding of fever patients with gruel made of barley and other farinaceous substances, to the exclusion of every other article of diet; yet gruels furnish few elements essential to the nourishment of a physical organization struggling against a subtle poison, and rapidly wasting with a burning fever, and starvation is the necessary result of a restriction to gruel diet.

There is no disease in which a waste of all the tissues of the body goes on so rapidly as in typhoid fever; and milk is an article of diet which furnishes the elements of nutrition necessary to repair this rapid waste, and there are not the objections to its use which there are against animal broths and gruels. Although there have been, and still are, in some quarters, strong objections against its use as an article of diet in fevers, recently it has been regarded with more favor, and those who have had most extended opportunities for testing its nutritive qualities have come to regard it as the only article of diet required by typhoid patients. In it we not only find all the elements required for repairing the rapidly wasting tissues, but they are in a condition to be most readily assimilated by the enfeebled digestive apparatus.

In order to make the milk more digestible, it may be di-

luted with lime-water. The lime-water is an antiseptic, and allays irritability of the stomach and intestines. The quantity of milk is not limited; the patient may take all his stomach will digest—usually patients will take from four to six quarts in the twenty-four hours.

After the patient has passed into the fourth week of the disease, you may find it necessary to administer cream and the yolk of eggs in connection with the milk.

Having considered the three most important things to be accomplished in the general management of typhoid fever, I now come to the treatment of the accidents of the disease.

DIARRHŒA.—I have told you that diarrhœa is one of the common symptoms of this fever; but it is one of which medical writers have taken special notice, and for the relief of which different means have been employed.

Let us for a moment notice the chain of phenomena of which diarrhœa is a link. The poison which produces this fever unquestionably has a specific action upon the intestinal glands and lymphatics. It is here that we find the characteristic lesions of the disease, and it is scarcely questioned that the typhoid poison, to a great extent, gains entrance to the system through these glands and lymphatics, and here produces the primary irritation. Following the irritation and inflammation of the follicles, other portions of the mucous membrane become involved, and we have a catarrhal inflammation of the mucous membrane of the intestinal tract. The necessary consequence of this is a diarrhœal discharge. Is this diarrhœa to eliminate the fever poison? Certainly not. It is simply an indication that these intestinal changes are going on; it is not due to the elimination of the typhoid fever poison, but to the inflammation which the fever poison has excited in the intestinal glands, and the subsequent intestinal catarrh. When the diarrhœa is present in the earlier period of the disease, it is better to let it alone. The question may be asked, will it not exhaust the patient? During the earlier period of the fever (the first and second week) the danger is very slight. It has been proposed to treat this diarrhœa, which makes its appearance early in the disease, with alka-

lies, bismuth, pepsin, etc. It is claimed, if these remedies be administered, diarrhœa can be prevented, or, if it already exists, that it can be controlled. Theoretically, I see no reason for employing alkaline remedies, for the diarrhœal discharges are always strongly alkaline, and, from clinical observation, I am convinced that bismuth, pepsin, etc., have little or no effect either in controlling the diarrhœa or in preventing the intestinal changes which produce it. When diarrhœa commences late in the disease (during the latter part of the third, or during the fourth week of the fever), it is of a very different character from that which occurs during the first and second weeks. Ulceration of the intestinal glands, and perhaps sloughing, has been established, and, in addition to the extensive local changes, there is a septic element which enters into the causation of the diarrhœa at this stage. Besides, the increased peristaltic action of the intestines, which attends the diarrhœa, favors an extension of the inflammatory processes to the peritoneum, especially that portion which covers the intestine, which corresponds to Peyer's patches. In view of these facts, the diarrhœa should be arrested or held in check. For the accomplishment of this, there is but one remedy which can be relied upon—that is, opium. My experience is against the use of astringents. If opium will not arrest it, you may expect little aid from astringents combined with opium as they are usually administered.

The use of opium is objected to by some, who claim that it diminishes the power of the heart's action; but in this disease, when administered in small doses, it seems to me to increase rather than diminish the heart-power. It is acknowledged that opium, more than any other drug, arrests the peristaltic action of the intestines; and that is what we wish to accomplish when diarrhœa is present during the third and fourth week of typhoid fever.

TYMPANITIS.—You will recollect that the tympanitis, which is sometimes so troublesome a symptom in typhoid fever, is due to gaseous distention of the intestines. Some assert that this gaseous accumulation is due to fermentative processes going on in the intestines; consequently that the

use of antiseptic remedies is indicated, such as muriatic acid, chlorate of potash, pepsin, etc. When this has proved a distressing symptom, I have usually found relief to be obtained by the application of turpentine stupes to the abdomen. Some claim that if turpentine be administered internally, from the beginning to the end of typhoid fever, that tympanitis and the intestinal changes which lead to it and to the diarrhœa are much less severe. I am confident that the turpentine treatment, as it is called, does not have the controlling influence over this fever which has been claimed for it; but I am also certain that it is our most reliable agent for the relief of the tympanitis.

INTESTINAL HEMORRHAGE.—Hemorrhage from the bowels in typhoid fever (as I have already stated) is a serious accident, and may cause death by producing a fatal exhaustion.

When it occurs early in the fever, usually it requires no treatment; but when it occurs during the third or fourth week, or after convalescence is apparently fully established, it must be arrested as promptly as possible.

The occurrence of severe intestinal hemorrhages may sometimes be prevented by keeping the patient in bed. A typhoid fever patient should not be allowed to get out of bed from the beginning of the attack until convalescence is fully established. Especially is this of importance if the case is a severe one, and attended by symptoms that indicate extensive intestinal lesions.

When hemorrhage from the intestines does occur during the third or fourth week of the fever, at once semi-narcotize your patient by the administration of opium in small doses at short intervals. Absolute rest of the body must be insisted on, the patient must not be turned on the side or moved in bed, and an ice-bag should be applied over the abdomen. I doubt if any good results can be accomplished by the use of astringents, either by enemata or by the mouth, as it is not known that they even reach the seat of the hemorrhage, although gallic acid and the persulphate of iron are usually recommended in cases of intestinal hemorrhage occurring in typhoid fever. If the hemorrhage is

profuse, it may be necessary to keep your patient under the influence of the opium for a week or ten days; in such cases the internal use of turpentine in connection with the opium will be found of service.

PERITONITIS.—When perforation of the intestine occurs, the case may be regarded as hopeless; death takes place usually within twenty-four hours; death occurs as the result of general peritonitis; no plan of treatment avails anything. If the peritonitis occurs without perforation, from the extension of the inflammatory process from the intestinal ulcers to the peritoneum, by bringing your patient rapidly into a state of semi-narcotism and holding him there for five or six days, you may prevent the extension of the peritonitis and thus save life. Such a case you are to treat in every respect as one of localized peritonitis.

After recovery from an intestinal hemorrhage or a localized peritonitis in typhoid fever, be exceedingly careful about the administration of cathartics or enemata; either may jeopardize the life of your patient. The bowels will move spontaneously after a time, even though the use of opium be continued, and no harm will follow should two or three weeks pass without a movement from them.

When the stomach is irritable, the hypodermic injection of morphine is preferable to opium administered by the mouth. This is given in sufficiently large quantities to paralyze the peristaltic movement of the intestines.

Bronchitis.—I have already stated that catarrh of the larger bronchial tubes is present in all severe cases of typhoid fever. No special treatment is required for its management; but, if the bronchitis becomes capillary, great relief will be obtained from the application of dry cups to the chest and the internal administration of carbonate of ammonia. Vapor inhalations will also be found of service in severe cases.

PNEUMONIA.—The pneumonia which complicates typhoid fever in nearly every case is lobular in character. The signs which indicate its occurrence are sudden rise of temperature, increased frequency of respiration, and the physi-

cal signs of localized pulmonary consolidation; cough and expectoration are rarely present.

Its occurrence is always an indication that stimulants should be administered. If they are being administered, they should be increased in quantity. To prevent or relieve the hypostatic congestion of other portions of the lung, which frequently accompanies pneumonic development, the heart-power must be increased, and the position of the patient changed.

LARYNGITIS.—For the relief of the laryngitis which occasionally complicates typhoid fever, a small blister may be applied on either side below the angle of the jaw, and the whole neck enveloped in a poultice. If these measures fail, and suffocation appears imminent, tracheotomy should be resorted to without delay.

SUBACUTE GASTRIC CATARRH, occurring as a complication during convalescence from the fever, can only be managed successfully by giving the stomach rest as far as possible, restricting the diet to a single tablespoonful of milk at a time, and applying hot fomentations over the epigastrium.

BED-SORES.—The severer forms of bed-sores are the most intractable complications we have to combat. Fortunately, the severer forms are much less frequently met with under the more recent plan of treatment; and, if they do occur, they are superficial and limited to small spots. Scrupulous cleanliness is one of the principal means for preventing their development. So long as there are no erosions, the parts should be frequently bathed in spirits of camphor, and the points of attack should be relieved from all pressure. If the sores penetrate the integument, they should be frequently washed with a weak solution of carbolic acid, or brushed over with equal parts of balsam peru and balsam copaiva and afterwards covered with dry lint or lint covered with vaseline.

The most unfavorable cases are those in which the point of pressure caused by the weight of the body becomes gangrenous. In such cases, by some a continuous warm bath is recommended. As soon as sloughing takes place, and

the parts separate, they should be dressed with lint saturated with balsam of peru and carbolic acid.

As has been already stated, diarrhœa is usually present in the early period of this fever; but sometimes there is constipation. The question arises, is the administration of cathartics ever admissible in typhoid fever? If so, what cathartic shall be employed? There is great diversity of opinion upon these points. One recommends the administration of rhubarb, another advises alkaline cathartics, and another would give calomel.

Quite diverse views are still held in regard to what the answer to this question should be. Recently, certain observers of extended experience have claimed that there is sufficient reason for the belief that a portion of the typhoid poison lodged in the alimentary tract may be expelled by the timely administration of cathartics, and thus the severity of the fever be mitigated and its duration shortened. Recent German writers claim that *calomel*, concerning the favorable action of which in this fever so much has been said and written, acts beneficially only as a cathartic. Those who favor the administration of cathartics recommend their use mainly during the first week of the disease.

On the other hand, equally competent observers maintain that the intestinal changes are augmented, and rendered more extensive by the action of cathartics; that the normal course of the fever is interfered with; and that in a large proportion of cases where intestinal and peritoneal complications occur, hypercatharsis has been induced at an early period of the fever by the administration of cathartics for the purpose of shortening its duration. My own experience leads me to exercise the greatest caution in the administration of cathartics in any stage of this fever. I am confident that the routine practice of administering purgative medicines in the early stage of typhoid fever can only be followed by a threefold injury:

First.—The patient is weakened.

Second.—The local intestinal lesions are increased.

Third.—Perforating peritonitis is more liable to occur.

The administration of cathartics as an eliminative procedure has neither reason nor experience to sustain it.

Before speaking of the management of the convalescence of typhoid fever, I will make a few general remarks on the use of anodynes for the relief of certain troublesome nervous phenomena.

I have stated to you that among the earliest, most frequent, and often most prominent nervous symptoms in this fever is headache, but it is seldom very violent or of long continuance.

Should it be severe, not readily relieved by fomenting the forehead and temples with warm water, or should it give place to active delirium, and other severe nervous disturbances, the question presents itself, shall anodynes be administered? If you decide to use them, the most reliable of this class of remedies is opium.

Usually, the condition of the pupil of the eye will serve to indicate to us whether opium shall or shall not be administered. A contracted or "pin-hole" pupil may be considered to contra-indicate its use, though there are exceptional cases in which opium acts favorably, notwithstanding this condition of the pupil.

Opium should be given with great caution whenever signs of cyanosis are present. In all cases of typhoid fever, it is safer to administer opium in small and repeated doses than to venture upon the administration of one large dose.

There are other anodynes which you will sometimes find of service, such as hyoscyamus, chloral, and the bromides. I would caution you against administering too large doses of chloral; the desired effect can generally be produced by ten or fifteen grains. If the first dose fails to relieve, a second may be administered at the expiration of two hours. This remedy is said to have a special value in quieting the active delirium, which is sometimes so troublesome, but my own experience in its use has not been favorable. When anodynes have failed to give relief to typhoid fever patients, who have been delirious and somnolent for days, they will sometimes become quiet and fall asleep immediately after the free administration of stimulants. Those

cases in which the nervous symptoms are due to an anæmic condition of the brain, associated with a weak heart and a flagging circulation, are most likely to be benefited by the use of stimulants. In those cases in which subsultus becomes very marked and there is a general tremor, jactitation, and restlessness, I have seen most happy effects produced by the use of hypodermic injections of sulphuric ether. I would use, as an average quantity, four drachms given in injections of one drachm each, in different places.

The same watchful care should be taken of a typhoid fever patient during convalescence as during the active period of the fever.

The number of typhoid patients who die during convalescence is proportionally large. Frequently this is due to the fact that the physician has laid down no strict rules to be observed as to diet and exercise, and frequently from the non-observance of such rules when they have been given.

The diet of fever patients during this period should be carefully watched. Allow your patient to eat frequently, but only small quantities of food should be taken at a time, so that the gastric juice secreted by the enfeebled stomach may be sufficient for its complete digestion. All indigestible articles of food, and those which furnish a large amount of waste, should be strictly forbidden. An apparently insignificant disturbance of the stomach, a slight vomiting, or a moderate diarrhœa occurring during the period of convalescence should be regarded as dangerous, for any one of these may induce a subacute gastritis, or lead to intestinal perforation and a fatal peritonitis. It is obvious that while the intestinal ulcers are healing, much mischief may be done by improper diet.

Notwithstanding the cravings of the patient's appetite, the diet must be restricted to such articles as milk, cream, gruels, jellies, and animal broths. Solid food must be strictly forbidden, especially meats, vegetables, and fruits. If diarrhœa is present during convalescence it is far safer to restrict the patient to milk and cream. All exercise, except simply walking around the sick-room, should be prohibited. I have had patients convalescing from typhoid fever sink

rapidly after a long ride, or after indulging in some violent and fatiguing physical exercise. It is of the greatest importance that this class of patients should keep in the recumbent or semi-recumbent posture until the cicatrization of the intestinal ulcers is completed, which in some instances does not take place for two or three weeks after convalescence is well established. If convalescence is slow, small doses of quinine, iron, and cod-liver oil are of service. They should be given after the patient has taken food.

When, during the period of convalescence, diarrhœa is persistent, the patient should be kept in bed, and some of the vegetable astringents, such as catechu, hæmatoxylon, may be employed.

In many cases it is important that you should take the evening temperature for at least two weeks after the commencement of convalescence, for by its range you will be able the more accurately to determine the exact condition of your patient.

When convalescence is delayed, so that at the end of four or five weeks the patient has not regained strength, change of air is indicated.

LECTURE VIII.

YELLOW FEVER.

Morbid Anatomy.—Etiology.—Symptoms.

THIS morning I will commence the history of the second in the list of miasmatic-contagious fevers, namely, YELLOW FEVER.

This fever has received its name from a yellow discoloration of the skin, which is a part of its clinical history.

The term, *yellow fever*, has been generally adopted by American, English, French, and German writers, and it is not necessary to mention the long list of obsolete names which have been applied to this disease by different writers.

MORBID ANATOMY.—We find that the anatomical changes which take place in the different organs and tissues of the body during the course of this fever, in some respects are similar to those which occur in miasmatic and contagious fevers, allying the disease more or less nearly to each of these classes of fever.

Although these different types of fever have many points of resemblance in their anatomical lesions, as well as in their general history, each has its own distinguishing characteristics which mark it as a distinct and specific disease.

The characteristic lesion (if we may so call it) of yellow fever is to be found in the liver. This organ is not much increased in size, but there is a striking and uniform change in its color. Sometimes it is of the color of fresh butter,

sometimes of a mustard color, and sometimes the color of coffee and milk, or chocolate color. In most instances this change occurs throughout the entire organ; occasionally, it is confined to one lobe, or to a small portion of a lobe. With this change in color there is a diminution in the quantity of blood in the liver, so that it contains less blood than normal. It has a dry appearance, is softer than normal, breaking down readily on firm pressure. When a section is placed under the microscope, it will be seen that there has been infiltration of the hepatic cells with oil-globules. In fact, all of the liver cells are more or less filled with oil-globules. Sometimes the change is a granular one, the nuclei of the cells have disappeared, or become obscured; in other instances, the entire liver cells have filled with large oil-globules, but the form of the cells has not changed.

This change has received the name of acute fatty degeneration. In its gross appearance, as well as in its minute anatomical changes, the liver resembles the fatty degeneration of the liver of rum-drinkers. Besides this, there is no change of any importance observed in the liver in yellow fever, except it may be slight extravasations of blood upon its surface, rarely in its substance.

MUCOUS MEMBRANES.—You will find the mucous membrane of the intestinal track, as also that of the larynx, the seat of a more or less severe acute catarrh. The vessels of the mucous surfaces, especially the veins, will present a turgid appearance; and so intense is the hyperæmia that at points they will present a varicose appearance. If there is a uniform congestion throughout the entire extent of the intestinal track, you will notice here and there little blood extravasations or ecchymotic spots. The whole track contains a greater or less quantity of fluid blood. Frequently the mucous membrane of the stomach is found thickened, reddened, and softened, sometimes with quite extensive blood extravasations. The contents of the stomach correspond to matters vomited during life, which I shall more fully describe under the head of symptoms.

HEART.—The heart is soft and flabby, lighter in color than normal, and will be found to have undergone degen-

erative changes similar to those which take place in its muscular tissue in typhoid fever. These changes undoubtedly do not depend upon high temperature, for a very high temperature is rarely present in yellow fever. The normal outline of the heart is lost, and it breaks down readily on firm pressure. The more severe the fever, and the longer its duration, the more extensive will be the parenchymatous degeneration. The pericardium usually contains one or two ounces of blood-stained serum. Partially organized clots are found in the heart cavities; these often extend for some distance into the vessels. They are the result of a slowing of the circulation from feebleness of the heart power, and, in most instances, are formed just prior to death, although they are not the cause of death.

LUNGS.—Usually the lungs are the seat of hemorrhagic infarctions. In fact, you will rarely make an autopsy upon one who has died of yellow fever without finding infarctions in the lungs, and sometimes they will be quite numerous. Diffused pulmonary apoplexies often occur, which may involve a large portion of a lobe. Under such circumstances the lung tissue will be broken down and occupied by large blood-clots. Spots of ecchymosis will also be found under the pulmonary and costal pleura.

KIDNEYS.—The kidneys are always more or less increased in size. This increase is due to swelling of the cortical substance, which is the seat of a more or less extensive fatty metamorphosis. It is a true parenchymatous nephritis, in which the fatty stage is very rapidly reached. You will find the uriniferous tubules crowded with oil-globules; in some places the tubes are denuded of epithelium; in other places they are filled with broken-down epithelium, which is undergoing a fatty and granular change. The pelvis of the kidneys is frequently the seat of acute catarrh, and evidences of catarrhal inflammation may be found along the ureters and in the bladder. The mucous membrane of the bladder will also be found to be the seat of punctate ecchymoses. In fact, in all the mucous surfaces of the body large and small ecchymoses are found.

BRAIN.—The brain and its membranes, as well as the spi-

nal cord, present no marked change. They are often hyperæmic, and are frequently the seat of punctate extravasation.

SPLEEN.—The spleen is but slightly, if at all enlarged, is of a darker color and of a softer consistency than normal.

SKIN.—The skin varies in color from a bright yellow to a dark orange. It may be the seat of ecchymoses or of large extravasations.

BLOOD.—There is one other important lesion of this disease, namely, the changes which take place in the blood. There is nothing characteristic about them; they are similar in character to those which take place in the blood in typhus and typhoid fever, although more extensive than in either. The blood coagulates much less rapidly and much less perfectly than normal blood. This loss of coagulating power may be due to a diminution in its fibrin, or to a loss of coagulating power in the fibrin. These changes were noticed by the earliest writers on this disease. The blood is changed in color, being darker than healthy blood. The blood-globules, instead of retaining their rounded outline, have their edges serrated and break down. There is no free pigment, such as is found in the different forms of malarial fever.

Blood taken from yellow fever patients rapidly undergoes ammoniacal changes.

Some of the pathological lesions of yellow fever very closely resemble those of relapsing fever. In both we find similar changes in the blood and a tendency to hemorrhages.

ETIOLOGY.—To the student of the literature of this disease, there is no part of its history so uncertain or so confusing as that of its etiology. Equally competent observers widely differ, and often hold diametrically opposite views in regard to it.

In our own city, some very bitter monographs have been written by medical men holding antagonistic views in regard to the causation of yellow fever.

I shall endeavor briefly to state well-authenticated facts concerning its causation, as far as possible making no mention of mere theories.

Under this head, the first question that presents itself is, In what localities does the fever prevail?

It is rarely met with north of 40° north latitude, or south of 20° south latitude. It prevails much more frequently on the western than on the eastern hemisphere, and in certain portions of Europe and America than in Africa. It is almost exclusively confined to commercial seaports, and is sometimes circumscribed to very narrow limits within those seaports. A certain amount of moisture, either on the surface or in the substance of the soil, is necessary for its production. There must also be present decaying animal and vegetable matter. For the production of the miasm which causes malarial fevers vegetable decomposition is sufficient, but for the development of yellow fever, both animal and vegetable decomposition is necessary. A high temperature is necessary to its development. The average temperature for the twenty-four hours must be above 77° F.

The period of the year during which yellow fever prevails depends upon climate and temperature. In the United States, it has usually appeared in July or August, and disappeared upon the first frost. The great epidemic of yellow fever in New York City, in 1795, began early in August, and disappeared about the middle of October.

Undoubtedly, this fever is indigenous in certain localities. There are certain seaports along our southern coasts, and certain islands of the sea, where it is developed whenever the necessary atmospheric conditions are present. Especially is it a disease of hot climates, and, in localities that are subject to it, it is more likely to prevail in very warm and wet than in cold and dry seasons. It may be endemic or epidemic. Sporadic cases are of rare occurrence, even in localities where it is indigenous. Some races more than others are subjects of this fever. The African race is most exempt from it.

A prolonged residence in a district where yellow fever is indigenous renders an individual less liable to contract the fever. Possibly a person may become acclimated to the

disease. Having once had the disease is a partial, though not complete, protection against a second attack.

North-westerly winds seem to arrest, while south-easterly winds seem to favor its development. In other words, when south-easterly winds are prevailing, the epidemic spreads and increases in severity, while, if the wind changes to the north-west, its progress is arrested. Whenever the temperature falls below the freezing point, no matter how pestiferous a region may have been, nothing more need be feared from the spread of the disease.

These are some of the conditions which are necessary for the development and spread or arrestation of yellow fever.

Now, the question arises, What is the nature of the poison that produces the fever? Is it a miasm or a contagion? There can be no question but that it is a poison in many respects similar to that of typhoid fever, which can be conveyed in some way from one individual to another, or rather that, when certain atmospheric conditions are present in connection with animal and vegetable decomposition, the introduction of the specific yellow fever poison is followed by its rapid reproduction. When it has been so reproduced, it may be received into the human system and give rise to morbid processes, attended by certain clinical phenomena which are characteristic of this disease. Thus far chemical and microscopical research has afforded no positive information in regard to the nature of the yellow fever poison, but there can be no question as to the existence of such a distinct and specific poison, and it would seem, from the conditions necessary to its development and the manner of its conveyance, that it is in some respects of the nature of a miasm, and in other respects that of a contagion. You may have yellow fever, remittent fever, and typhoid fever, all prevailing at the same time in a locality, yet each of these three diseases will run its individual course, and no one will lapse into another.

The question now comes to us, *Is yellow fever contagious?* There are three leading doctrines upon this point.

First.—The doctrine of *unqualified contagion*, which

attributes to the disease an absolute and unqualified contagious character.

Second.—The doctrine of *non-contagion*, which maintains that the disease is never transmitted directly from one person to another.

Third.—The doctrine of *contingent contagion*, which teaches that the disease cannot be conveyed from one individual to another, except in a yellow fever atmosphere; that is, when yellow fever is prevailing in any locality, in that locality it may be transmitted from one person to another.

After carefully studying the recorded observations and weighing the statements of the advocates of these different doctrines, I can unhesitatingly state to you that the majority of those who have had the most extended opportunities for studying this disease, deny its contagious character, and very strongly advocate the doctrine of non-contagion.

Some German writers claim that the germ in yellow fever, as in typhoid fever, cannot be conveyed directly from the sick to the healthy, but that it must first be deposited in decomposing animal and vegetable matter, and that wherever animal and vegetable decomposition is going on, there are present the conditions necessary for the rapid reproduction of the yellow fever germ. One thing is certain, that whenever yellow fever prevails as an epidemic, there is present animal and vegetable decomposition. The disease prevails only where men are crowded together, as in ships, and around the docks and wharves of seaports, and in the filthy streets and habitations of such localities.

In some few instances, evidence exists that yellow fever breaking out in the hold of a vessel has been circumscribed to certain portions of the hold by free ventilation, not a case occurring save within certain well-defined limits, within which ventilation was impracticable.

It would therefore seem that yellow fever can be produced only when the atmosphere has become loaded with emanations from animal and vegetable decomposition, to which must be added the specific yellow-fever poison before the fever can be propagated from the sick to the healthy.

While the advocates of the doctrine of non contagion are

positive as to the non-contagious character of yellow fever, they are equally certain that it is a portable disease, that is, that it can be conveyed from one locality to another by means of clothing, merchandise, and in the holds of vessels. They also believe, when yellow fever poison is thus introduced into healthy localities which are suited by temperature and the presence of animal and vegetable decomposition to its reproduction, that it rapidly and repeatedly reproduces itself, and in this way epidemics of yellow fever may be developed in localities which are usually free from the disease. Consequently, it is a disease which should be guarded against in any seaport by a vigorous quarantine.

How long yellow fever poison may retain its vitality is not yet positively determined, but that the period is a very long one there can be no question.

One may visit a locality where yellow fever is prevailing and remain in it for a considerable time, and not convey the poison in the clothing beyond the boundaries of the district where the disease is prevailing. In order to the conveyance of the poison beyond these limits, it is necessary that the clothing become so saturated with the poison that it will not become neutralized when exposed to the air of a non-infected district.

I have briefly stated to you all of the important well-ascertained facts that bear upon this vexed question. In conclusion, it may be stated that with the written history of the disease before one, there is not sufficient evidence to lead to the acceptance either of the doctrine of contagion, or of contingent contagion. It seems to me there need be no fear of contracting the disease by visiting those sick with yellow fever in a yellow fever district, unless such visits are very much prolonged. The poison of yellow fever, as met with in the holds of vessels, sometimes is so concentrated that a very short exposure is sufficient to overwhelm the nervous system, and give rise to very urgent nervous phenomena, which are soon followed by the development of the fever, and from such exposure it is possible to convey the poison in the clothing.

The length of the period of incubation varies from twelve

hours to four or five days ; it is claimed by some that this period of incubation may extend over a period of several weeks. When the exposure is followed in a few hours by the fever, the yellow fever poison must necessarily be very concentrated.

SYMPTOMS.—The development of yellow fever may or may not be preceded by premonitory symptoms, such as headache, pain in the limbs, and loss of appetite. If these symptoms are present, they are by no means characteristic of the fever. In nearly every instance the disease is ushered in by a distinct chill ; in no disease, unless it may be puerperal fever, is a chill so invariably an ushering in symptom as in yellow fever. While apparently in the most perfect health, while at work, or even while asleep, patients will be seized with a slight or severe chill, and immediately become seriously ill, taking their beds in the most disheartened manner.

You will remember that I stated to you that there were both mild and severe types of typhoid fever, and that they differed only in degree, not in kind ; so also is the case in yellow fever, and you must remember this fact when considering the symptoms of this fever.

The outline of the clinical history is very nearly the same in a mild as in a severe type of the fever. Following the chill or rigor which ushers in the attack, there is supra-orbital headache, pains in the back and limbs, which are especially severe in the calves of the legs. The countenance is flushed, the conjunctiva congested ; the eye has a peculiar lustre and a staring look. The temperature rises rapidly, and reaches 102° F. within a few hours after the chill. The temperature in yellow fever varies very much in different cases. In some cases it never rises above 102° F., while in some severe epidemics it has been recorded as high as 110° F. Such a temperature is very seldom reached. By the end of the second day the temperature usually reaches its maximum height, which rarely is higher than 105° F. In this country, according to records made, the temperature has rarely risen higher than 104° F. This fever is not characterized by so high a range of temperature as is

met with in almost all the other varieties of fever. From the second to the fourth day the temperature variations are slight, and do not amount to distinct remissions. By the fourth day, if not before, the temperature falls very rapidly, so that in twelve hours the normal standard may be reached; usually, however, it does not fall below 100° F. This fall constitutes a distinct remission. This period of remission may last from a few hours to two or three days, after which time the temperature again rises, and rapidly reaches 104° F., or even rises higher; then it remains stationary from twenty-four to forty-eight hours, after which time it falls to the normal standard, where it remains until convalescence is established.

In accordance with the temperature variations, the disease may be divided into three stages: a *first* stage, or stage of invasion; a *second* stage, or stage of remission; and a *third* stage, or stage of exacerbation.

Some writers have divided the disease into a febrile stage, or stage of exacerbation, a passive stage, or stage of remission, and a stage of collapse.

LECTURE IX.

YELLOW FEVER.

Symptoms (continued).—Differential Diagnosis.—Prognosis.—Treatment.

THIS morning I would invite your attention to the farther study of the symptoms of yellow fever. I have stated to you that in the majority of instances this fever is ushered in by a distinct chill ; usually, this is not prolonged ; following the chill there is a rapid rise in temperature, which, by the third or fourth day, reaches its maximum height, from 103° F. to 107° F. This rise in temperature may be accompanied by dryness of the surface, or the surface of the body may be bathed in a profuse perspiration. Sometimes, after the chill has subsided, there is an unnatural coldness of the surface, and there seems to have been no rise in temperature, but the thermometer in the rectum registers 104° F. or 105° F.

PULSE.—The pulse in yellow fever is never accelerated in proportion to the rise in temperature. It rarely becomes as frequent as in other forms of continued fever, seldom reaching more than 110 beats per minute. In quite severe cases it may only reach 100, and in the milder cases it may not be accelerated more than five or six beats. It has a peculiar character ; many writers term it a “ gaseous pulse.” It is easily compressed and has an uncertain volume and character. This peculiarity of pulse is an element of differential diagnosis.

EYE.—The eye is suffused, and the conjunctiva becomes congested quite early in the disease. The appearance of the countenance in severe cases has almost uniformly been regarded as diagnostic of this disease. The eyes are red and watery, and the conjunctivæ are so intensely congested that the eyes resemble two balls of fire, while the face has a dusky, deathly hue; these give to the countenance a remarkable expression of dejection and distress.

TONGUE.—The tongue is early covered with a thick white coating, except at its tip and edges, which are red, and in fatal cases, towards the close of life, sometimes the tongue becomes dry, brown, cracked, and fissured, resembling the tongue of typhoid fever. There is loss of appetite, and from the very onset of the disease there is more or less nausea and vomiting.

VOMITING.—Nausea and vomiting may be regarded as among the most constant and characteristic symptoms of yellow fever. They come on soon after the chill, and continue throughout the entire course of the fever. At first the matters vomited are simply the contents of the stomach, then they become yellowish or greenish in color, are fluid, and have an alkaline reaction. There is nothing about the matters vomited that is characteristic of yellow fever. If the vomiting subsides without any other changes in their character, it is quite evident that the case is going on to recovery. In the fatal cases the vomiting continues until a few hours previous to death, and in some cases until the hour of death. In a large proportion of these cases there is finally developed the striking and well-known *black vomit*, which has been regarded as characteristic of this fever, and which by some is supposed to occur only in this disease. This peculiar vomiting may occur upon the second or third day of the fever, but more commonly it does not come on until thirty-six or forty-eight hours previous to death, or not until the day of death. It undoubtedly occurs more frequently in yellow fever than in any other disease, but it differs in none of its constituents from a similar material which is sometimes vomited in other diseases. A microscopical examination of the black vomit

shows it to consist of pigment matter in the form of fine granules, aggregated non-granular masses, and globules which resemble blood-globules. In addition to this coloring matter there are found epithelial cells from the mucous membrane of the stomach, lymphoid cells or white globules which have undergone degeneration, and serous fluid. The pigment material is due to changes produced by the action of the gastric secretions upon the blood that has escaped from the walls of the vessels of the mucous membrane of the stomach into its cavity. The action of this gastric secretion upon the red blood-globules is such as to permit the escape of their coloring matter in the form of granules or small rounded masses. The same change will occur whenever blood escapes in small quantities by a capillary hemorrhage into the cavity of the stomach. Although this may occur in other diseases, yet when it does occur in yellow fever, it should be regarded as a grave symptom. The vomiting is projectile in character, and in this respect is peculiar to this fever.

The bowels are usually constipated. If diarrhœal discharges are present, generally they are of a dark color, and frequently contain fluid blood, as there is the same tendency to capillary hemorrhage from the mucous membrane of the intestines as from the stomach.

URINE.—The changes in the urine are regarded by some as diagnostic. Early in the disease it has an acid reaction. As soon as bile is present in the urine its reaction becomes alkaline. By some this alkalinity is regarded as an evidence of commencing convalescence, but there is no reason for such an opinion, for this change in the urine might be expected as soon as bile becomes one of its constituent parts, but it is not due to any peculiar action of the yellow fever poison.

Albumen has been found in the urine in all fatal cases, and early in the attack, it has been found present in moderate quantity, in all severe cases, but as the disease progressed it became more or less abundant according to the severity of the fever. *Entire suppression of urine* is of frequent occurrence in severe cases, and no symptom, not even the

black vomit, is so unfavorable as the complete suppression of urine. A patient with black vomit does not necessarily die, but complete suppression of urine is almost invariably followed by a fatal termination.

Under the head of the morbid anatomy of this disease were described the kidney lesions which account for the suppression of urine; for if the kidney changes are extensive, it is impossible for these organs to perform their function, and death is the necessary result, for acute uræmia is added to the fever poisoning.

Delirium is rarely present, but when it occurs it is wild in character, and it is marked by a constant desire on the part of the patient to get away from some impending danger. Usually the mind is clear, but a peculiar apathy takes possession of these patients, and they often lie in a state of complete collapse, with shrunk features, entirely indifferent and unconcerned as to their condition.

JAUNDICE.—The yellow color of the skin, which is so prominent and constant a symptom of yellow fever, usually does not appear until about the third day of the fever. It is first noticed about the eyes, but soon extends over the entire body. Some have maintained that this discoloration of the skin is a true jaundice, due to a retention and reabsorption of bile, in the same manner as we have acute jaundice following an obstruction of the gall duct. Those who do not accept this doctrine maintain that the gall ducts are not found obstructed, hence there is no reason for the retention and reabsorption of the bile.

The true etiology of the jaundice in yellow fever may be stated as follows: the yellow fever poison is introduced into the circulation, and produces its specific changes in the blood; that is, the red globules are destroyed within the circulation, the hæmatine in them is changed into pigment matter, and a staining of the tissues of the body follows, which is a real hæmatogenous jaundice.

Admitting these blood-changes to have taken place, we find a ready explanation of many other changes which take place in this disease. The blood-globules to a great extent are destroyed, and in consequence the blood loses its vital-

izing power. This induces defective nutrition, and leads to fatty degeneration of the liver cells and the renal epithelium. The walls of the capillary vessels become enfeebled, to which is added the qualitative alterations in the blood itself, and these lead to hemorrhagic extravasations in various parts of the body which mark the progress of this disease.

It is of importance that you remember that in yellow fever, about the third or fourth day, sometimes within twenty-four hours from the commencement of the attack, the temperature rapidly falls, so that in twelve hours it may reach its normal standard. In the majority of instances it does not fall below 100° F., and there is no distinct intermission, but a decided remission. The pain in the head and back now subsides, the patient is in every way very much improved, and you may consider him convalescing. Yet, in a day or two, there may be a return of all the febrile and other distressing symptoms which were present in the early period of the fever; after these have continued for twenty-four or forty-eight hours, usually convalescence is established; especially is this the case when recovery is to take place as soon as the remission is established. In such cases, with the remission, the pain in the epigastrium, the vomiting, and the yellow discoloration of the skin all begin to subside. The patient is now able to take nourishment, and with the occurrence of these symptoms, if the surface has been dry, a slight moisture appears, and the patient soon passes into a state of convalescence.

On the other hand, the vomiting may continue, and the black vomit appear; the distress and burning in the epigastrium may become more and more severe; there is greater restlessness, tossing, and agitation; the albumen in the urine is more abundant; the urine becomes more and more scanty, until finally complete suppression occurs, and coma and death follow.

Some epidemics are marked by a predominance of one class of symptoms and some by the predominance of another class, so that it is difficult to give a history of this fever which shall accord with all its different modes of de-

velopment. Consequently, there have been many varieties of yellow fever described, such as the comatose, the algid, etc. Strictly speaking, these so-called varieties are simply variations in the clinical manifestation of the disease produced by the degree of poisoning, and by some peculiarity in the atmospheric conditions under which it prevails.

Some epidemics are much more fatal than others, and the ratio of mortality is much less during the latter part than during the early part of an epidemic.

At the present time, there seems to be little question but that the immediate cause of death in all severe epidemics of yellow fever is due to uremia. The yellow fever first produces its changes in the blood, which leads to such glandular changes, especially of the kidneys, as arrest glandular functions, and a secondary blood-poisoning is the result.

Some writers have described a period of collapse. It is true that a condition of collapse not unfrequently occurs, but it is nothing more than a period of commencing death.

DIFFERENTIAL DIAGNOSIS.—Yellow fever has been confounded with malarial fever, relapsing fever, and with acute atrophy of the liver. Under ordinary circumstances the diagnosis of yellow fever is not difficult, yet there are certain types of malarial fever which are especially liable to be mistaken for it.

Some writers have even gone so far as to maintain that the so-called bilious-remittent is only a modification of yellow fever. At the present day, it has been fully established that each is a distinct type of fever. The following are the points of differential diagnosis between them:

First.—The character of the prevailing disease, the region in which it prevails, and the manner of its endemic or epidemic development. Yellow fever prevails in seaports, remittent fever in inland towns. Yellow fever *is*, remittent fever *is not* portable.

Second.—The difference in the manner of invasion of the two diseases, the difference in the range of temperature, the projectile character of the vomiting in yellow fever, and its non-projectile character in remittent, the peculiar character

of pulse in yellow fever, as well as the almost characteristic expression of the countenance, is quite sufficient to distinguish it even from the so-called yellow type of remittent fever. Then the difference in the anatomical changes, and in the effect of quinine in the two diseases is very striking. There is a yellow discoloration of the skin in both diseases, but it appears earlier and is more intense in yellow than in remittent fever. The presence of an enlarged spleen would lead to the diagnosis of remittent rather than yellow fever.

Relapsing Fever.—At the first appearance of this disease in a new locality it may be confounded with yellow fever. You will be led to a correct diagnosis by studying the etiological relations of the two diseases. *Relapsing fever is, yellow fever is not*, propagated by contagion. Then, the almost typical range of temperature in relapsing fever furnishes a marked distinction between it and yellow fever. In the former if yellow discoloration of the skin is developed, it does not come on until late, generally not until the relapse. An enlarged spleen is the rule in relapsing, and the exception in yellow fever. Hemorrhage from the mucous surfaces may occur in both these types of fever, and there can be little question but that the blood-changes are very similar in kind, but not in degree, in these two forms of fever. During the past two years, in the wards of Bellevue Hospital, in two instances, has acute yellow atrophy of the liver been mistaken for yellow fever. If an accurate history of the cases could have been obtained, doubtless the mistake in diagnosis would not have been made.

In yellow atrophy of the liver, as well as in yellow fever, there is jaundice with fever, and vomiting of a black material accompanied by suppression of urine; but the history of the development of the two diseases and the gradual but steady diminution in the size of the liver in yellow atrophy, while in yellow fever the organ rather increases than diminishes in size, is sufficient for a diagnosis.

The difficulties which attend the differential diagnosis of yellow fever are often very great; in fact, sometimes it is impossible to make a positive diagnosis. For example, some

of the crew of a ship coming from an infected port become jaundiced, have hemorrhage from the mucous surfaces, accompanied by fever of a remittent type ; if these patients have previously suffered from intermittent fever, attended by an enlargement of the spleen, it will be almost impossible in the earlier cases to decide between so-called bilious-remittent and yellow fever.

Prognosis.—The average duration of yellow fever is six days ; sometimes it destroys life in three days. The prognosis greatly varies in different epidemics. The highest recorded ratio of mortality which I have been able to find is one death in every three cases.

Some writers have claimed that more than one-half the cases are fatal, but upon a careful examination of statistics I find they give no such percentage of death. In some epidemics the fever is of so mild a type that only a very few cases terminate fatally, perhaps one in fifteen or twenty.

A consideration of the following conditions is of importance in making our prognosis :

The severity of the invasion of the fever. The intensity of the febrile excitement. The early appearance of the yellow tinge of the skin and the intensity of the jaundice. The greater the severity of the period of invasion, the higher the range of temperature ; the deeper the jaundice, and the greater the amount of albumen in the urine, the more unfavorable is the prognosis. If the quantity of albumen diminishes, the patient is advancing toward recovery ; if it increases, a fatal termination is indicated.

The elements of a favorable and unfavorable prognosis may be briefly stated.

The *favorable symptoms* are a slow pulse, a slight rise in temperature, a quiet stomach. Streaks of blood during the latter stage of the fever are not regarded as indicating danger, especially if the blood-corpuscles are entire. Albuminous urine without casts is not of serious import. Under all circumstances, a copious secretion of urine must be regarded as a favorable symptom.

A recent residence in a temperate climate will enter very largely into the chances of recovery from yellow fever.

The *unfavorable symptoms* are: a high temperature, a red tongue, an irritable stomach, intense pain in the head, scanty urine, containing albumen and casts, black vomit, a faltering articulation, and difficulty in protruding the tongue. A streak of blood in the early vomit indicates great danger, especially if the blood-globules are broken down. The intensity of the jaundice, and the fact that the patient has recently suffered from an attack of yellow fever, render the prognosis unfavorable.

In a large number of cases you will find great difficulty in giving a positive prognosis. The presence of the "black vomit" and an entire suppression of urine render a case almost hopeless, as has already been stated. Recovery after the occurrence of "black vomit" is more frequent than after suppression of urine. In mild and in severe cases the period of convalescence is in proportion to the duration of the disease. In some cases it is not fully established until two weeks after the cessation of the febrile symptoms. Complete recovery does not take place in some cases until five or six months after the commencement of convalescence.

There are no certain sequelæ of yellow fever. Cellulitis and abscesses are spoken of by some writers, but they are by no means constant.

TREATMENT.—Before considering in detail the treatment of yellow fever, I would say a few words concerning its prophylaxis. The prophylactic measures for the most part are included under the general head of quarantine regulations. It is possible by strict quarantine to prevent the introduction of yellow fever into any district or seaport where it is not indigenous. It is not necessary that I should enter upon a discussion of those quarantine regulations which have been found most successful in preventing the introduction of this disease; these come rather within the province of State medicine. If you find yourself in a region visited by an epidemic of yellow fever, you may escape it by removing beyond the limits of the infected district. If you are compelled to remain within the limits where the epidemic is prevailing, avoid everything which is regarded

as a predisposing cause of the disease. Under such circumstances most observers regard the sulphate of quinine, taken daily in moderate doses, as a prophylactic agent.

The details of the treatment to be employed when the disease has once established itself are very unsatisfactory ; perhaps there is no disease the treatment of which is more unsatisfactory. Medical men widely differ as to the most effectual means to be employed in controlling or mitigating the severity of the fever. Physicians in India, and American physicians who have come in contact with this fever, treat it very differently. Within the past few years there has been a marked change in the views of American physicians in regard to its treatment.

The remedial agents which have been most extensively used are mercurials, bleeding, stimulants, and quinine. It is very difficult to accurately estimate the relative value of these different agents, for this reason, there are certain forms of this fever in which no treatment avails anything, the patient receives his death-blow at the very onset of the fever. On the other hand, there are forms of so mild a type that the patient is almost certain to recover. Hence the great uncertainty which attends any plan of treatment, and the unreliableness of statistics in regard to its effects. Under all plans of treatment there are many deaths and many recoveries. I have already alluded to the four leading plans of treatment which have been resorted to for the management of this fever, namely, the mercurial, the blood-letting, the stimulant, and quinine plan. The plan now most generally adopted is the expectant, or, as it is called by some, the diaphoretic.

At one time blood-letting was very extensively practised in the treatment of yellow fever, one hundred and eighty ounces of blood have been drawn from the temporal artery at a single bleeding. The most experienced and intelligent physicians, with the largest opportunities for observation, have abandoned this plan of treatment, which fact is sufficient argument against it. The same is true of the mercurial plan of treatment ; now mercury is only employed as a cathartic at the very commencement of the fever.

The stimulating plan of treatment has also fallen into disrepute. It was found that the administration of stimulants during the active period of the fever was not followed by good results.

Again, our most competent observers unhesitatingly declare that quinine has no controlling power over the fever.

Let us pause a moment and consider what are the indications as to treatment.

The great danger in yellow fever is that the kidneys will fail to perform their function.

What more sensible plan of treatment than that which contemplates relieving the kidneys from excessive work? Here is an opportunity for the use of diaphoretics, and a certain amount of cathartic medicine, not to use them to such an extent as to produce exhaustion, but so far as to afford as much relief as possible to the kidneys.

At the commencement of the attack counter-irritation over the region of the kidneys is undoubtedly of great service.

The plan of treatment now most generally recommended and adopted is, as soon as a patient is taken with yellow fever, in addition to the application of counter-irritants over the region of the kidneys, to administer ten grains of calomel combined with ten grains of quinine. Why the quinine is added to the calomel I do not know. Keep up a moderate diaphoresis. At the same time administer lime-water and milk, which is said to have greater control over the nausea and vomiting than any other means which have been employed.

It has been recommended that the surface should be bathed with some alkaline lotion, on the theory that alkalines applied to the surface have a controlling influence over the vomiting. There are no reliable facts to sustain this theory.

In severe cases, during the fever, there is usually nausea, great restlessness, with tossing and rolling of the head. In order to quiet this uneasiness and jactitation some have proposed the use of chlorodine, others the administration of chloroform, but all have protested against the use of opium,

because of the kidney lesions, insisting that by the use of opium in any form we ran the risk of causing additional disturbance of the function of the kidneys.

I regard this restlessness to a great extent as due to the effect produced upon the nerve centres by the urea in the circulation, and believe that all these nervous manifestations can best be controlled by the hypodermic use of the sulphate of morphine.

Perhaps it may be worthy of mention that a physician living in the West Indies has recently quite successfully treated cases of yellow fever by administration of carbolic acid in doses of one and a half to two grains every two hours. It is claimed that the carbolic acid given in this way arrests the changes in the blood produced by the yellow fever poison. I should question very much if carbolic acid has any such power.

As the course of this fever is very rapid, it is of the utmost importance to sustain the vital powers as far as possible till the morbid processes come to an end. This is always difficult on account of the great irritability of the stomach—but as soon as the stomach is in a condition to receive food, you must endeavor to improve the composition of the blood by a most nutritious diet, combined with wine, quinine, and iron.

MALARIAL FEVERS.

LECTURE X.

MALARIAL FEVERS.

Introduction.

WHEN I began the history of fevers, you will remember that I divided them into three general classes, namely, the contagious, the malarial, and the miasmatic-contagious.

This morning I commence the history of those which are included under the head of *malarial fevers*. I pursue this course for the reason that I believe you will be better prepared to study contagious fevers after you shall have become familiar with the malarial. The different varieties of malarial fever are like different branches of the same tree; they have many things in common, yet differ from each other so widely in the phenomena which attend their development, that they may be regarded as distinct diseases. They have a common origin in a poison which has received the name of *miasm*.

All varieties of these fevers depend upon one and the same poison, which is subject to certain variations in quantity. The concentration of this poison determines the severity and, to a certain extent, the type of the fever. It is possible to arrange the different types in a progressive scale, from the mildest to the most severe, beginning with the simple intermittent and passing on to the most severe type of pernicious fever. The extent of the morbid processes, and the rapidity with which they are developed, depend upon the intensity of the malarial poison, the length of time the individual has been under its influence,

and, to some extent, upon individual idiosyncrasies. Many theories have been advanced as to the nature of this *miasm* or malarial poison. By some it is regarded as gaseous in its nature; others believe it to be a living vegetable organism; and, again, others think it is a specific poison, having no tangible, chemical, or microscopical constituents.

No one of these theories, nor of the many others which at different times have been advanced, have been sustained either by facts or by reliable chemical or microscopical analysis. Thus far we have *no positive knowledge* in regard to its true nature, but we do know something of the circumstances which are necessary for its production and the laws which regulate its development.

First.—There must be a certain amount of vegetable matter, either on the surface or in the substance of the soil, where the malarial poison is generated. It is not necessary that the quantity be large, but a certain amount is a necessity.

Second.—A certain amount of moisture must be on the surface or in the substance of the soil; it need not be excessive; but some is indispensable.

Third.—A certain average degree of temperature is necessary for its production. It cannot be developed below an average temperature of 58° F. for the twenty-four hours, and will not prevail as an epidemic unless the average temperature ranges as high as 65° F. for the twenty-four hours.

In regions where these fevers prevail, their type, form, and intensity, to a great degree, depend upon the height of the temperature.

As a rule, malarial fevers are endemic, rarely extending over large sections of country in the form of an epidemic. I will repeat, three things are known to be necessary to the development of miasm or malarial poison, namely: *the presence of decomposing organic matter, a certain amount of moisture, and a certain average range of temperature.*

We also have some knowledge concerning the regions in which malarial fevers are most likely to prevail, and which seem most favorable to the development of malarial poison.

First.—Marshes are especially favorable to the develop

ment of this poison, and may generate it for an indefinite period. The Pontine marshes have been malarial for more than two thousand years. Yet all marshes are not malarial; their power to generate the malarial poison varies with the amount of water they contain. Where there is an abundance of water, malarial fevers are rare; when they are covered only by a thin sheet of water, and are exposed to the direct rays of the sun, malarial poison will abound. Marshes that have dried up are especially favorable to the development of this poison, yet as soon as heavy rains submerge the previously parched surface, the power to generate the poison is for a time diminished or entirely arrested.

Scattered here and there over our own continent are districts which have been malarial ever since the white man has held possession of them; whether such was the case in earlier times, our history is too uncertain for us to determine.

As a rule, salt-water marshes are especially free from malaria, but when salt and fresh water become mixed in the marsh, as, for instance, on the New Jersey flats, you have the most favorable conditions of marsh for its abundant development. Those marshes resting on a substratum of sand are far less malarial than those resting on limestone, clay, or mud.

There are marshes in the higher latitude of our own and other States which often, during the heat of summer, become dry, yet no malarial poison is generated (although during the day the thermometer may reach 90° F.); for this reason, that during the night the atmospheric temperature falls below 50° F.

There are some quite extensive marshes in which apparently every condition for the development of malaria exists, and yet none is generated. We cannot account for this fact, unless we accept the theory that the ozone which is claimed to be present in such marshes arrests or prevents its generation.

“Damp bottom-lands” that are exposed to an annual overflow, such as are found along the southern shores of

the Mississippi River, are as fruitful as swampy regions in the generation of this poison.

Second.—Another condition which seems to favor the development of malaria is the upheaval of new alluvial soils, such as obtain when new lands are first brought under cultivation. This same state of things also occurs throughout the middle and southern portions of this State, and in the New England States.

Where railroad excavations are made, malarial fever is very frequently developed.

In this city, while the so-called "Fourth avenue improvements" were being made, the entire region along the avenue was rendered highly malarious by the excavations. Such excavations bring decomposing vegetable matters to the surface; these, under the influence of heat and moisture, generate miasm.

The fact that fevers of this type appear in regions previously free from them, as soon as these conditions favorable to their development exist, is confirmed by the testimony of many careful observers.

Third.—Regions otherwise non-malarial may have malarial poison brought to them by the waters of rivers which have their source in, or flow through, malarial districts.

Examples of this kind are found along the banks of our Western rivers, where are developed some of the most pernicious types of this fever; while in places only a short distance from these rivers it is unknown.

This can be accounted for, if we accept the theory that malarial poison has been transmitted through waters having their source in, or running through, malarial districts.

Fourth.—Non-malarial regions may be rendered malarial from poison transmitted by the wind.

There has been considerable discussion as to whether this poison can be transmitted in such a manner, and if it can be, to what distance. I find no reliable account of its transmission over a greater distance than four and three-quarter miles.

Malarial fever broke out in the crew of a ship, which was anchored just four and three-quarter miles from shore where

this fever was prevailing. No cases were on board when the anchor was cast, nor did any of the crew go on shore. So long as the wind blew from the ship towards shore, the crew remained well, but when the wind changed its direction and blew from the shore towards the ship, within six days from the time of change, cases of well-developed malarial fever appeared on board. This seemed to prove conclusively that the fever was brought to the ship by the wind.

The wind may also carry malarial poison up along the sides of mountains, to an elevation of one thousand feet; some writers say no higher than six hundred feet.

American writers give no account of its being carried higher than six hundred feet, while some German writers give well authenticated cases, which show that it must have been carried to the height of one thousand feet.

I have thus far called your attention to some of the more important conditions which are necessary to, or seem to favor, the development of this malarial poison. You have seen that certain of these conditions are absolutely necessary for its production. I have also noticed most of the conditions which render its development more active.

I will now briefly consider some of the circumstances which are inimical to its production.

First.—High latitude. In this country malarial poison is not generated in higher latitude than that of Quebec. The limit of its development is 63° north latitude, and 57° south latitude. Between these two parallels of latitude, both on the eastern and western hemispheres, malarial fevers may be developed; the nearer the approach to the equator, the more severe the type. They do not prevail over the entire region embraced between these parallels of latitude, but it is possible for them to be developed at any point where the altitude is not too great.

Second.—High elevation is another condition inimical to its development. As a rule (as I have already stated), it is not generated above an elevation of one thousand feet above sea level.

There are, however, some remarkable exceptions to this

rule. We find recorded cases of malarial fever which have been developed upon plateaus among the Pyrenees, at an altitude of 5,000 feet. I have already referred to the fact that malarial poison is much more readily developed in marshes which have a clay or lime-stone bottom, than in those which have a sandy or porous substratum. Among the Pyrenees, there is a marsh which has a clay bottom, and there malarial poison is developed which is very persistent.

Third.—Drainage is another means which diminishes, and in certain conformations of soil entirely destroys malarial generation. In the majority of marshes, this generation can be arrested or prevented by free drainage. Yet there are marshes upon which millions have been expended in drainage, which still remain pestiferous.

Perhaps it is possible to drain the Jersey flats so as to render them non-malarial in their character, but it is hardly probable that this change can be effected, for they have a clay bottom, and contain both salt and fresh water, conditions which I have stated are most favorable to malarial generation. Years of labor and large expenditures of money have been bestowed upon the Pontine marshes to render them non-malarial, yet they are as pestiferous as they were two thousand years ago.

Fourth.—Cold is a powerful agent in arresting malarial generation. I care not how pestiferous a region may have been, if only for one night the temperature fall below the freezing point, nothing more need be feared in that region from malaria, until the average temperature shall have again reached 60° F. This law holds in all malarial districts. In these districts, after the temperature has fallen below the freezing point, persons may have the fever, but it is the result of previous poisoning.

Again, the generation is less rapid and the poison is less virulent during the day than at night. This is the uniform testimony of those who have seen most of, and written most on malarial diseases. It is also almost universally conceded that malarial districts are most pestiferous during months when the atmosphere is hot and dry, with little or

no wind, especially when this state of atmosphere has been preceded by long, heavy rains, and that the virulence of the poison is greatly diminished as soon as fresh, strong winds clear the atmosphere.

I have called your attention to the most prominent laws which seem to govern the production of this poison, as also I have endeavored to bring before you those conditions which favor, as well as those which hinder or prevent its development. The question now arises, How does malarial poison gain entrance into the human body ?

The most reasonable view is that this is effected through the respired air. Certain facts seem to show that it may be introduced through the intestinal tract with the food and water. There seems to be scarcely a doubt but that it may be taken into the stomach with foul drinking-water. Accepting this view, in certain localities it has come to be the practice to add whiskey to the drinking-water to destroy the poison, but there is no reason for the belief that whiskey has any such power.

When this poison has once been introduced into the circulation, it undoubtedly has the power of reproducing itself, hence the entire system is affected. From this fact, which must be regarded as well established, those who regard this poison as a living organism, claim that these organisms may reproduce themselves indefinitely, but their existence has never yet been demonstrated. It has also been claimed that certain races are more exempt than others from malarial fever, also that there are idiosyncrasies of constitution which render certain individuals exempt from diseases of this type, for in districts where these fevers prevail there are persons who never have the fever.

It seems to me that this exemption, both in races and individuals, is due to the greater physical power of the individual, which enables him to resist these noxious atmospheric influences.

In a district where malarial influences prevail, the weak and anæmic are the most liable to be attacked, and all those influences which tend to lower vitality, and to render feeble the powers of resistance, must be regarded as special predis-

posing causes. A strong man may resist for a long time, while the old man and the child very quickly succumb to the influence of the poison. Women are more susceptible than men to its influence. You can no more account for the fact that one person can take in large doses of malarial poison without being effected by it, while another is affected by a very small quantity, than you can account for the fact that one individual can take large quantities of alcoholic stimulants without showing any signs of intoxication, while a very small quantity will intoxicate another individual, supposing, in both instances, the individuals to have apparently an equally vigorous constitution.

Some claim that when an individual has been poisoned with malaria, complete recovery never takes place; others claim that even with the worst cases recovery is possible. My own experience leads me to believe that when an individual has once suffered from malarial poisoning, he is much more susceptible than one who has never been so poisoned. For instance, an individual suffers from one or more attacks of intermittent fever, and then removes from a malarial district, if that person again enters a malarial region, he is much more likely to suffer from malarial fever, however slight the poisoning may be, than if he had never suffered from its effects. Some unknown physical change has taken place which renders him a fit subject for malarial manifestations upon the slightest exposure.

This brings us to the doctrine of the latency of malarial poison in the human body. This is an interesting and at the same time a very obscure subject.

That there is a period of incubation, or rather that a certain time elapses between the exposure and the development of malarial fever, seems to be a settled question. For, often a long, always a short period elapses before newcomers in malarial districts have their first attack of the fever; sometimes the poison remains latent until after they have removed from the district. It is on this basis, the latency of the malarial poison, that the relapses can be accounted for, which occur in those who, having lived in a malarial district, remove and remain in a non-malarial one.

This reawakening of the malarial poison may depend upon a variety of causes, such as taking cold, over-fatigue, sudden changes of temperature, etc., etc.

Whether an individual who has once been thoroughly poisoned with malaria can ever become entirely free from its influence, is still an unsettled question.

From my own observation, I am convinced that it is impossible to bring one wholly from under the influence of the poison while he remains in a malarial district, though he may become exempt from its influence (without the reawakening causes already mentioned, taking cold, etc., etc.), if he remains beyond the malarial belt.

Undoubtedly, you have often heard it stated that an individual may become so acclimated as to resist malarial influences, and live for a long time in a malarial district without suffering any evil effects from it.

There can be no question but that those living in such districts suffer less from the acute manifestations of the poisoning than do new-comers. But the truth is, those changes, which we call chronic malarial affections, are constantly going on in those who are supposed to be acclimated.

The comparison still holds good in reference to those addicted to the use of alcohol. We might say, they are becoming acclimated to its use. The first dose a person takes may make him drunk, but after a time repeated and larger doses fail to produce this effect. Malaria acts like any other poison: after a time the system reaches a certain degree of tolerance.

This tolerance of malaria, or immunity from its manifestations, amounts to nothing more than the accommodation of the system to its prevailing influence.

Let the acclimated person, as he is called, be taken sick with any active form of disease, such as diphtheria or pneumonia, and it usually proves fatal, not that there is anything unusually severe in the diphtheria or pneumonia which brings about the fatal termination, but death is due to the fact that the system is charged with malarial poison.

There is another point in this connection concerning which I wish to say a few words.

It has been claimed by very intelligent and careful observers that phthisical developments are prevented by malarial poisoning. After having carefully investigated this subject, I am convinced that the effect of the poison on the human organization is to predispose it to phthisical developments. The milder climate and the less frequent changes in temperature in the malarial regions accounts for the fact that there is less phthisis in those regions than in the cold, non-malarial regions. The malarial districts in the northern portion of the temperate zone have the highest death rate from phthisis. If we accept the fact that the larger number of cases of phthisis are catarrhal in their origin, and that catarrhal pneumonia is more likely to be developed in those who are broken down from the prolonged influence of malarial poisoning, you will be prepared to understand how chronic malarial poisoning predisposes to phthisis. In quite a number of instances I have traced the beginning of phthisical development to this cause.

There are many other points of interest closely connected with this subject of malarial poisoning, but which have no special connection with the class of diseases which we are about to study.

LECTURE XI.

SIMPLE INTERMITTENT FEVER.

Morbid Anatomy.—Etiology.—Symptoms.—Differential Diagnosis.—Prognosis.—Treatment.

I HAVE spoken of the origin of malarial fever, and of certain known facts concerning the development of the malarial poison, and to-day will commence the history of this class of fevers. First in order is simple intermittent fever.

Like typhoid fever, simple intermittent fever is met with in all parts of the world, although the region of its development may be said to be limited by 63° north latitude and 57° south latitude. Within these parallels it is the more prevalent the nearer you approach the equator.

MORBID ANATOMY.—The anatomical changes which take place in this fever are few and require only a passing notice. In regard to the blood-changes we are without any reliable chemical or microscopical data. We find none of those changes in the blood which are present in the more severe forms of infectious disease, neither do we find those which are present in the pernicious type of malarial fever, such as pigmentation and marked diminution in the red globules. If the fever has continued for a long time there may be slight diminution in the number of the red globules and a decrease in the fibrin of the blood; but these changes, to a great extent, are due to the high temperature which attends its paroxysms. The only constant pathological lesion of simple intermittent fever is congestion of the internal organs. The spleen and liver are always more or less en-

larged, but the enlargement is due to simple hyperæmia; no structural changes occur in these organs until the intermittent paroxysms have been often repeated, and the malarial poisoning has been of long duration. There is also more or less hyperæmia of the kidneys and the mucous membrane of the intestines, but it is not attended by any signs of gastric or intestinal catarrh. As yet no one has been able to prove that any structural change takes place either in the nerve tissue or in any other tissue of the body; nor from the structural or functional disturbances that occur during the fever, has any one been able to find a satisfactory answer to the question, why it is a paroxysmal and not a continued fever? By some German writers it is claimed that during a paroxysm of the fever white blood-globules are very rapidly developed; but the question arises, how is this to be demonstrated? I have never seen a post-mortem examination on one who had died during a simple intermittent paroxysm, and have never heard of such a death unless the patient had some intercurrent disease. As I have already stated, all the appreciable lesions of simple intermittent are those of hyperæmia.

ETIOLOGY.—At my last lecture this subject was brought to your notice. All agree that simple intermittent fever is due to malarial poisoning, and that the poison is introduced into the body either through the lungs or through the intestinal tract.

Whatever tends to depress the mental or physical powers of an individual renders him more susceptible to malarial influences, and consequently these depressing influences must be regarded as predisposing causes. Among these may be included intemperance, exposure to night air, excessive fatigue, bad hygiene, and a long list of like debilitating causes.

SYMPTOMS.—This fever is a paroxysmal disease, of different types, according to the period of time between the paroxysms.

The *first*, and most common, is the *quotidian* type, in which the paroxysm occurs every day, and there is an interval of twenty-four hours between the paroxysms.

Second, you have the *tertian* type, in which the paroxysm occurs every third day, with an interval of forty-eight hours between the paroxysms.

Third, you have the *quartan* type, in which the paroxysm occurs every fourth day, with an interval of three days or seventy-two hours between the paroxysms.

These are the regular and more common types of intermittent fever. Medical writers make mention of other types, which, although irregular, are unquestionably modifications of those already mentioned. Among these is what is described as *double quotidian*, in which two paroxysms occur daily. Usually one paroxysm is severe, the other mild: the severer one generally occurs in the morning, the milder in the afternoon or evening. There is also a *double tertian*, in which a paroxysm occurs daily, but it differs from the quotidian, as the paroxysms that resemble each other occur at intervals of forty-eight hours. For instance, the paroxysm of to-day is characterized by the occurrence of a severe chill and mild fever; to-morrow it is characterized by a short chill and severe fever; the following day there occurs the severe chill and mild fever, as on the first day.

Some writers describe a form of intermittent fever in which the paroxysm occurs on the seventh, fourteenth, twenty-first day, etc., with an interval of seven days between the paroxysms.

The types most frequently met with are the quotidian, tertian, and quartan.

In the quotidian variety the paroxysm occurs in the morning, in the tertian it occurs about noon, while in the quartan it occurs in the afternoon or evening. The duration of the paroxysm varies with the type of the fever. In the quotidian it lasts from eight to ten hours, in the tertian it lasts from six to eight hours, in the quartan from four to six hours.

There are many exceptions to these rules, but it is a question whether we would have them if the disease was permitted to run its course without treatment.

PAROXYSMS.—A paroxysm of intermittent fever has three

stages, namely, the *cold* stage, the *hot* stage, and the *stage of sweating*. In most cases these are easily distinguished the one from the other.

In the true type of intermittent fever we have regular intervals between the paroxysms of fever.

Let us notice some of the phenomena which attend one of these paroxysms.

After the patient has suffered for a certain length of time with pain in the head, a sense of languor, and some nausea, he passes into the cold stage.

COLD STAGE.—His passage into this stage is first marked by a sensation of coldness along the back, which soon extends to the extremities, and an uncomfortable sensation of coldness gradually creeps over the entire body. The skin becomes shrivelled, the finger ends and lips become blue, the face is pale, the eyes are sunken, chills rapidly follow each other, the teeth begin to chatter, any voluntary motion is attended by trembling, until finally, as one chill after another in quick succession passes over the body, the patient's teeth chatter so that it can be heard some distance from the patient, and there is a shaking of the entire body.

The surface of the body becomes rough, the blood seems to recede from it, and the skin assumes the appearance described as *goose-skin* or *cutis anserina*. The temperature of the surface of the body is lower than normal, but if you place the thermometer in the axilla or under the tongue you will find that the temperature has reached 104° F. or 105° F. The voice of the patient is weak and husky, the respirations are rapid, short, and sighing, but the mind remains clear. The urine is increased in quantity, and paler than normal, and there is frequent desire to empty the bladder. Usually these symptoms are present from half an hour to two or three hours; the length of time depends upon the severity of the case.

After the cold stage has continued for a longer or shorter period, the patient begins to have flashes of heat alternating with the chilly sensations. Usually these are first felt at the extremities, but they rapidly extend over the whole body, and the *hot stage* is established.

HOT STAGE.—The skin, in this stage, is no longer shrivelled, but becomes red, swollen, and turgid, and there is a recession of the blood from the central organs to the surface of the body. That the temperature is elevated can be ascertained simply by laying the hand upon the surface. If, however, you place the thermometer in the axilla, in most cases you will find the temperature has reached 106° or 107° F. The thirst is very much increased. The comfortable sensation which the patient experienced while passing from the cold to the hot stage has given way to great restlessness and uneasiness, the patient tossing from side to side, with face flushed, and eyes red and fiery. Sometimes herpetic vesicles appear about the mouth. The heat and thirst become intense, the tongue becomes dry, the carotids pulsate, the radial pulse becomes firmer and more rapid than in the cold stage, and nausea is now a marked symptom. It may have been present in the cold stage, but in the hot stage nausea and vomiting become the prominent symptoms. As a rule the symptoms of this stage last from half an hour to two hours. In exceptional cases they may continue for a much longer time. As I have already stated, the ordinary duration of a paroxysm of a quotidian intermittent is from eight to ten hours; that of a tertian, from six to eight hours; and that of a quartan, from four to six hours. It is possible, especially in those forms of malarial fever in which the poisoning is intense, for the hot stage of a quotidian to continue twelve hours. There is no condition in which, for the time, you have more intense fever than in the hot stage of intermittent fever. The urine, which, during the cold stage, was abundant and of pale color, now becomes highly colored and scanty. Not unfrequently it is almost suppressed during the hot stage. Complete suppression of urine occurs only in the pernicious type of the disease. When the fever has continued for a longer or shorter time, a slight moisture appears upon the forehead which gradually spreads over the entire body, and the patient becomes bathed in a profuse perspiration. He is now in the *sweating stage*.

SWEATING STAGE.—As this stage comes on the former

restlessness and uneasiness passes away, and a feeling of comfort comes to the patient as the perspiration makes its appearance. The temperature rapidly falls; the pulse rapidly diminishes in frequency and force; the pulsation of the carotids ceases; the face assumes its normal appearance; the congestion of the conjunctiva disappears; and the patient rapidly passes from a high state of fever into one in which he falls asleep, and awakens after a period ranging from one to three hours, with a sense of exhaustion.

Interval.—During the interval between the paroxysms at first the patient may feel perfectly well, but if there is a frequent repetition of the paroxysms, there will very soon be a marked loss of vitality; he becomes pale and feeble, and all the symptoms of malarial cachexia are present. There will be more or less of a jaundiced hue to the skin, enlargement of the spleen and liver, and pigmentation of the tissues. It is true that many paroxysms of simple intermittent may occur before any such general disturbance of the health of the patient manifests itself; yet, in the interval between the paroxysms, we cannot call the patient's condition one of perfect health.

Usually, in the quotidian type, the day previous to the development of the first paroxysm, unnoticed by the patient, there is a slight rise in temperature, perhaps from $99\frac{1}{2}^{\circ}$ F. to 103° F. At the same time he experiences a sense of lassitude, and is disinclined to make any exertion, either mental or physical. The temperature commences to rise in the morning, and by noon it has reached its maximum height; then it begins to fall, and by evening it may have fallen to nearly its normal standard. Thus the course of the temperature is quite characteristic, and may be summed up as a rapid ascent, a short and intense stationary period, and critical defervescences constituting the paroxysms, with a perfectly normal temperature in the interval. The following day another rise in temperature will be noticed; now the rise does not occur in the morning, but after mid-day, perhaps so late as in the evening. Usually in the quotidian type of intermittent fever the highest temperature is reached a little earlier each day; if it is reached a

little later, you may be certain that the fever is being modified or controlled by treatment. We have what are called *anticipating* and *postponing* paroxysms. When the paroxysm comes on a little earlier each day, it is called *anticipating*, and indicates that the fever is not being controlled; when it comes on at a later hour each day it indicates the fever is being controlled, and is called a *postponing* intermittent.

The types of intermittent fever which occur most frequently in temperate climates are the quotidian and the tertian. With us the quotidian is most frequent. In those who have suffered repeatedly from intermittent fever, the disease is liable to run an irregular course, the paroxysms occurring on irregular days, and with irregular intervals. In children this fever shows certain deviations from the ordinary course. The paroxysms may be ushered in by convulsions, or by a period of stupor. Children rarely have the distinct chill. After a period varying from ten minutes to half an hour, we have the hot stage of regular intermittent fever coming on, with all its attendant phenomena. The intermissions are rarely complete. The child loses his appetite and flesh, becomes irritable, and has a pale, waxen look, and suffers from gastric and intestinal disturbances, and the intermittent very soon lapses into a remittent.

DIFFERENTIAL DIAGNOSIS.—The differential diagnosis of simple intermittent fever is never very difficult. There are only two diseases which are liable to be mistaken for it, namely, remittent fever and pyæmia. It is readily distinguished from remittent fever, for in remittent fever there is never a complete intermission, whereas in intermittent there is always a period in which there is no fever. A careful thermometrical observation for twenty-four hours settles all question in regard to it. In remittent, the temperature, when at its lowest point during the remission, is one or two degrees higher than normal, while in intermittent the temperature reaches the normal standard during the intermission.

There is also a regular development of the paroxysm in intermittent, which does not occur in remittent. In remit-

tent usually you have but one chill, while in intermittent a chill precedes each paroxysm of fever.

The diagnosis between intermittent fever and pyæmia is also readily established. In pyæmia, there is no complete intermission in the fever and no regularity in the time of its occurrence, or in the severity of the paroxysms. In both diseases you have chills, fever, and sweats, but in pyæmia the chill is short; rapid shivering is followed by a prolonged and very high fever, and this is followed by profuse sweating. The sweating of intermittent is never so profuse as that of pyæmia, and in the latter disease there is no regularity in the development of the phenomena, while in intermittent, the nature of the paroxysms, and the time of their occurrence, can be predicted with great certainty. The principal element in the clinical history of pyæmia is a steady, high temperature, without any intermission. When the sweating comes on the temperature may fall one or two degrees, but it never approaches the normal standard, and there is never a distinct intermission.

It is much more difficult to make a differential diagnosis between pyæmia and remittent fever than between pyæmia and intermittent. Hereafter this will be more fully considered. The same thing may be said in regard to the hectic fever of phthisis.

PROGNOSIS.—The prognosis in simple intermittent fever is good. If continued for only a short time, there will be no tissue changes to prejudice the life of the patient.

The possibility of the development of malarial cachexia must enter into the prognosis. When this occurs the case is more than one of simple intermittent fever; there is enlarged spleen, enlarged liver, and pigmentation of tissues.

TREATMENT.—The treatment of intermittent fever is divided into that for the paroxysm and that for the interval. The treatment for the paroxysm, in most cases, is simply to render the patient as comfortable as possible while passing through its various stages. At one time it was proposed to tourniquet the limbs, so as to prevent congestion of internal organs, and thus arrest the paroxysms.

Again, it has been proposed to apply cold to the surface

of the body, for the purpose of giving a shock to the nervous system, and in that manner to arrest the paroxysm. To accomplish this, by covering the surface of the body with sinapisms, in order to irritate the cutaneous surface, has also been proposed. Some have claimed that if an individual is brought fully under the influence of alcohol the regular development of a paroxysm can be prevented. Again, it has been claimed that opium, given in full doses at the usual time for the recurrence of the paroxysm, has power to prevent it.

Experience does not lead me to accept any of these statements. It is true that, in some instances, a sudden shock to the nervous system may prevent the development of an intermittent paroxysm when the paroxysms have become a habit.

If there is anything in the entire list of means (either remedial or hygienic), that I have named, which has power to prevent the full development of a paroxysm, it is opium. When this is administered hypodermically, early in the cold stage, it will diminish the severity of the cold and hot stages. Whether, in the treatment of the milder forms of intermittent fever, the combination of opium with quinine is advisable, is still an unanswered question, though it seems to me that in such cases much comfort can be afforded, and the patient be much less injuriously affected by the paroxysm, if opium be administered in moderate doses.

Patients with intermittent fever should be kept in bed during the entire paroxysm, however mild it may be. During the cold stage, cover them with blankets, surround them with bottles of hot water, and let them drink freely of hot water. All these means will hasten the hot stage of the disease. During the hot stage, the extra clothing and external heat should be gradually removed, and cold instead of hot drinks should be administered. If nausea and vomiting are present in this stage, you will find that opium, administered hypodermically, affords great relief.

When the patient reaches the sweating stage, let him alone; within a few hours he will be entirely relieved, and in a state of convalescence. The question now arises, What

treatment shall we adopt during the interval to prevent the occurrence of another paroxysm? If possible to prevent it, never allow a patient to have a second intermittent paroxysm; for if the system once becomes accustomed to these paroxysms, they will be repeated upon the slightest provocation. You will frequently find this to be the case with persons who for a long time have not been subjected to malarial influence, and yet upon the least nervous excitement or fatigue will have a paroxysm.

Let me impress upon you to prevent, if possible, the occurrence of a second paroxysm of intermittent fever.

The great remedy at this time is the *sulphate of quinine*. Skillfully used, it is all-powerful to accomplish this result. How and why it arrests the development of these paroxysms I do not know. We simply know the fact. Our knowledge of its antiperiodic power is purely empirical. There is much difference of opinion as to the mode in which it should be administered. In commencing the treatment of a case of intermittent fever, after the occurrence of the first paroxysm it is always safe to assume that the fever is of the quotidian type. At least thirty grains of quinine should be administered between the termination of the one paroxysm and the hour when another is to be expected. The first dose of ten grains should be given towards the close of the sweating stage, and twenty grains about two hours before the time of the expected paroxysm. If possible, give the quinine in solution. If there should be sufficient irritability of the stomach to cause the rejection of the quinine, it may be administered hypodermically, or by enema. Three grains administered hypodermically has about the same antiperiodic power as ten grains administered by the stomach. If you succeed in preventing the occurrence of a second paroxysm you have accomplished much for your patient.

Having prevented the occurrence of a second paroxysm, it is important that a moderate degree of cinchonism should be maintained for a number of days, by the daily administration of quinine in moderate doses. About two hours before the time of day at which the first paroxysm occurred, from ten to fifteen grains of quinine should be daily admin-

istered. You must not now permit your patient to pass entirely from under your observation. Direct him to visit you *one month from the date of the first paroxysm*, for, although he may not have had a fresh malarial exposure, there will be a strong tendency at this time to a repetition of the paroxysm, and it is of importance that your patient at that time should be again brought fully under the influence of the quinine. If it is possible for your patient to remove from a malarial district you will be almost certain to prevent a second paroxysm.

If, however, you do not see your patient in his first paroxysm, and he lives in a malarial district, sulphate of quinine, administered in the manner I have just recommended, may only prevent for a time the return of the paroxysm, and even complete cinchonism may fail to control it. You should now very carefully examine the case, in order to ascertain if there is not some condition present which interferes with the antiperiodic action of the quinine, such as hepatic or splenic hyperæmia. When careful percussion shows that the liver and spleen are increased in size, even after the administration of full doses of quinine, you will often find that the administration of full doses of calomel with the quinine will increase the antiperiodic power of the latter, and thus diminish the percussion area of these organs.

Occasionally, when full doses of quinine combined with calomel have failed to prevent a recurrence of a paroxysm, I have noticed an unusual excitement attending its development, and believing from this circumstance that, owing to individual idiosyncrasies, the malarial poison had a more than usual irritating effect upon the nervous system, I have accomplished the desired result by administering full doses of opium with the quinine. In fact, if the patient is of a highly sensitive, nervous organization, I never allow a second paroxysm to pass without administering a full dose of opium before the time when its return is to be expected. In all those cases which are called obstinate, ascertain why you have failed to control the disease by the use of quinine.

I rarely have administered arsenic in simple intermittent fever. If I fail to control the fever with quinine, after I have reduced splenic and hepatic congestion, controlled nervous irritability, and increased nutrition by the administration of iron and the moderate use of stimulants, I never succeed with arsenic. In some of the chronic forms of malarial manifestation, I have found arsenic of great service, but never in simple intermittent fever.

Other means employed in the treatment of this fever will be spoken of in connection with pernicious fever.

MASKED INTERMITTENT.—In this connection I would invite your attention for a few moments to a form of intermittent fever, which by some writers has been designated *masked* intermittent fever. For example, to-day a patient has a regular intermittent paroxysm, but to-morrow, instead of its recurrence, perhaps, he suffers from the most intense neuralgia. This neuralgia may have its seat in the intercostal or in the sciatic nerve, or perhaps more frequently in the frontal portion of the ophthalmic branch of the trigemini nerve. Some one nerve becomes involved and no other seems to be affected.

In some cases, an intense hemicrania takes the place of the paroxysm.

As a rule, these neuralgias have distinct intermissions, and so come to be regarded as masked forms of intermittent fever.

Instead of a neuralgia, your patient may have an attack of asthma, or an attack of indigestion. During the past year I have seen several cases of intermittent dyspepsia. The patient, after having had one or two distinct intermittent fever paroxysms, or perhaps only a slight chill, fever, and sweat, has suffered severely from indigestion, colicky pain in the bowels, and symptoms resembling those of peritonitis. Diarrhœa, dysentery, and sometimes hæmaturia and apparent suppression of urine, may take the place of a distinct intermittent fever paroxysm.

Again, your patient may have a single well-defined chill, or even two chills followed by most intense hemicrania, and then have no more for a long time, but sooner or later

he will have a well-defined intermittent paroxysm which will reveal the real nature of the disease.

Sometimes this form of intermittent fever instead of being a quotidian, a tertian, or a quartan, may be one in which the paroxysms are developed every sixth or seventh day. I might refer you to other types of this fever, which we might call masked intermittent, but which in their development do not present the regular phenomena of a fully developed paroxysm.

LECTURE XII.

SIMPLE REMITTENT FEVER.

Morbid Anatomy.—Etiology.—Symptoms.—Differential Diagnosis.—Prognosis.

THIS morning I shall commence the history of Simple Remittent Fever, the second in my list of malarial fevers.

This is a continued fever, with diurnal exacerbations. It is known by different names, such as Southern, Western, African, Continued, Bilious, Acclimative, and Remittent Fever.

The term, Remittent Fever, is more generally accepted, and the one which I shall adopt.

MORBID ANATOMY.—In many respects, the anatomical lesions of remittent fever resemble those of intermittent fever, yet there are certain points of difference with which it is important that you should become familiar. These differences are rather in degree than in kind.

Unquestionably, both these types of fever are the result of malarial poisoning; therefore, we may expect the same diminution of the red globules and the same changes in the fibrin of the blood in remittent that we have noticed in prolonged intermittents. Yet there are other changes in the blood, which we usually find present in the former, that are of quite rare occurrence in the latter, namely, the presence of free pigment-granules. These pigment-granules are met with in some of the pernicious forms of intermittent fever; but, in all cases of well-developed remittent fever, they are present at some time during the progress of

the disease. This pigmentation is due to the hæmatoidin which has its origin in the hæmoglobin which has been liberated from the blood-corpuscles within the blood-vessels, and then developed in the liquor sanguinis. This coloring matter may remain either within the blood-corpuscles, which, after a time, become transformed into pigment-granules, or remain free in the fluid portion of the blood, or infiltrate the adjacent cells and tissues. It may be transformed into granular or crystalline hæmatoidin.

The *spleen* is not so much enlarged in remittent as in intermittent fever, and the increase in size seems to be of a different nature. The enlargement is evidently the result of congestion, and the organ sometimes presents very nearly the same appearance as it presents in typhoid fever, except that there is more pigmentation present, which is rarely present in a typhoid spleen.

There are also structural lesions found in the liver, in the stomach, and in the intestines, which are not present in intermittent fever. The liver is not very much increased in size, and, in color, is of a bronze hue. The principal change is in color, which is uniform throughout its entire substance. This varies in degree in different types of the disease, and in different cases of the same type. The peculiar color is due to pigmentation of the liver tissues, and varies according to the amount of pigment deposited. Pigmentation may occur in other tissues of the body, but not to the same extent as in the liver. On a microscopical examination of the liver tissue, pigment is found throughout its entire structure—not only in the hepatic cells, but in the nuclei of these cells and in the walls of the blood-vessels.

This discoloration is of such uniform occurrence that it has been recognized in different countries and by different writers as the characteristic pathological lesion of remittent fever. Consequently you will find in your books that the "*bronzed liver*" is spoken of as the characteristic lesion of this fever. Occasionally you may have the same pathological lesion in intermittent and pernicious fever, but this is so seldom, and its presence is so constant in remittent fever,

that if you meet with it at an autopsy you may venture upon the diagnosis of remittent fever.

STOMACH.—You will find the mucous membrane of the stomach more or less congested, thickened, and softened. In this respect the disease is somewhat allied to typhoid fever. You will find similar changes also in the mucous membrane of the intestines; it is more or less congested, and presents very much the appearance seen when a moderately severe catarrhal inflammation is present. The Peyreian patches are usually enlarged, and quite frequently present the “shaven beard” appearance. In some cases there are ulcerations, not, however, as extensive or of the same nature as the ulcerative processes of typhoid fever. The mesenteric glands are not enlarged, and there is none of that granular infiltration in the glands so noticeable in typhoid fever. There is only a simple hyperæmia, entirely due to a catarrhal inflammation. Thus you notice in taking up the history of each of these fevers, that while each one is a distinct disease, we find many things that are common to all of them. There is in all some pathological change which seems to link them together.

The same changes may occur in the muscular tissues of the body, which are met with in typhus and in typhoid fever, and they are claimed by some to be the result of prolonged high temperature; yet in remittent fever the temperature rises higher than in typhoid, while these muscular degenerations are of rare occurrence, and less extensive when present. The more we study these fevers the more disposed, it seems to me, will we be to attribute these granular degenerations to something besides high temperature.

The most important characteristic change, and perhaps the only one, in all malarial fevers, is the change which takes place in the blood-globules.

ETIOLOGY.—The great predisposing and exciting cause of this fever is malarial poisoning. There can be no question but that the same malarial poison which gives rise to intermittent fever can produce a remittent fever. In other words, we have remittent passing into intermittent fever,

and intermittent passing into remittent fever. While it is possible for this to occur, as a rule the two diseases do not prevail in the same locality at the same time. Endemics of one form may occur and be followed by endemics or sporadic cases of the other form. In some localities remittent fever is almost the only form of malarial disease, intermittent fever only occasionally occurring.

There is probably no form of endemic disease, the geographical boundaries of which are more extensive than those of remittent and intermittent fever. With certain exceptions they may be said to encircle the earth by a broad belt, parallel with the equator, limited by 63° north latitude, and by 57° south latitude. The boundaries of this belt are quite irregular, now approaching the line of the tropics, now receding from it.

The remittent fever which occurs within the northern or southern limits of this belt is much less severe than that which occurs in the tropical regions. From the localities in which this fever prevails it would seem that a higher average temperature is required for its development than is required for the development of intermittent fever. In certain portions of this immense tract cases of remittent fever are never seen; especially is this the case at a distance from the equator, while in the tropical regions the places of exemption are comparatively few.

As I have already stated, a remittent fever during its convalescence may become an intermittent, and, conversely, an intermittent, either from new exposure to malarial influences or to the influence of high temperature, may become a remittent. From this fact the conviction is forced upon us that under differing circumstances both these types of fever may be developed from a common malarial poison. Usually certain atmospheric changes will have taken place to change the type of the fever. They rarely prevail endemically at the same time. For instance, intermittent fever may prevail early in the season, but as the season advances, and the temperature ranges higher, the fever which prevails will assume the remittent type.

Some claim that each of these two forms of fever has a

distinct malarial poison, but I believe the difference to be, not in kind, but in degree. There are certain circumstances which predispose one person more than another to an attack of remittent fever. For instance, those who go from a non-malarial district into one where remittent fever is prevailing are more likely than those who live in the infected district to have this fever.

Remittent fever is governed by the same laws in its development that govern the other forms of malarial fever.

It prevails along the banks of rivers; the miasm which produces it may be conveyed by the wind; it occurs in marshy regions where there is but little water. When the same localities in which intermittent prevails are exposed to a higher degree of temperature, remittent fever may be developed. These laws have already been sufficiently considered under the head of malarial poisons.

SYMPTOMS.—The ushering-in symptoms of remittent fever are usually more marked than those of any other form of continued fever.

The most constant as well as the most urgent of the premonitory symptoms is oppression in the epigastrium. This may be present for forty-eight hours, or even a longer time, previous to its development. There is also a certain amount of lassitude, nausea, and loss of appetite, and with these feelings uneasiness and perhaps pain in the head and limbs. There is very much the same feeling of general discomfort that precedes the development of typhoid fever. But the disease does not come on gradually, as does typhoid fever, but abruptly, usually with a chill. There is no question as to when the patient began to be sick. The cold stage is neither so complete nor so long continued as it is in intermittent fever or pneumonia.

It is of importance that you remember this peculiar feature. During the chill the thermometer will indicate a temperature two or three degrees above the normal. With the chill there is a most intense headache, pain in the back and limbs. Following the chill, there is fever, during which the temperature rises very rapidly. The fever increases in severity, and, within twelve hours from the time of its

commencement the temperature will have reached 105° or 106° F. As a rule, the chill is not of so long duration as the chill of intermittent fever, neither does it begin, like it, by creeping down the back and gradually extending over the body, but there is general coldness over the surface of the body at the very commencement of the chilly sensation. Again, there is not that tremulousness and shaking of the body, neither that chattering of the teeth, which is so frequently experienced in intermittent fever. In a few words, the chill of remittent is not so severe as that of intermittent fever.

As soon as the temperature commences to rise, the pulse is increased in frequency, and perhaps reaches 100 or 120 beats to the minute. The face becomes flushed, but not so intensely flushed as in the second stage of intermittent fever. The eyes are usually suffused, and the conjunctiva is somewhat congested. The patient is restless, tossing in bed, in the vain search of an easy posture. As the hot stage advances, nausea and vomiting are always present, and the sense of oppression in the epigastrium increases, which is not relieved by vomiting.

In making a diagnosis, remember that this disease is ushered in by a *chill*, followed by a *fever*, which is accompanied by nausea, vomiting, and great distress in the epigastrium.

We have nausea and vomiting occurring in intermittent fever, but it is not so persistent and distressing in character as the nausea and vomiting of remittent. Again, there is not the same amount of pain in the epigastrium, for in the febrile stage of remittent fever the patient suffers from it to such an extent that quite commonly it is the only thing of which he complains.

Before this, there has been a sense of oppression and perhaps pain in the epigastrium, but during this period the epigastric distress is very great, and is often accompanied by the most extreme tenderness upon pressure. The material first vomited simply consists of the contents of the stomach, next follows the vomiting of a greenish matter, and finally, in severe cases, even of simple remittent fever,

you may have a slight amount of black vomit. This resembles the black vomit of yellow fever. The quantity of fluid vomited is greater than the quantity taken into the stomach.

Vomiting of stringy mucus tinged with green is always present in remittent fever. Sometimes the patient's stomach rejects everything taken into it, and the vomiting is accompanied by terrible distress in the stomach, pain in the head, and general disturbance of the system.

At the commencement of the fever, usually, the bowels are constipated.

The symptoms thus described go on increasing in severity for ten or twelve hours, then you will notice a slight amount of perspiration upon the forehead. In a short time, it extends over the entire body, not profuse, but a slight moisture upon the surface. With the perspiration will be a fall of one or two degrees in temperature, and a fall of ten or twenty beats in the minute rate of the pulse. The thirst will diminish, the vomiting grow less, there may now be ability to retain fluids taken into the stomach, and the patient falls into a quiet, refreshing sleep, and is relieved from all the severer symptoms of the paroxysm. If, however, you will place the thermometer in the axilla, you will find that evidences of fever still exist, and although there has been a marked decline in temperature, it does not reach the normal standard. At no time is there a complete interruption; the fever is continuous. This is termed the period of remission. At the same time on the following day all the active febrile symptoms return, increased in severity, the range of temperature is higher, the gastric disturbance is more marked and severe, the countenance assumes an anxious expression, and all the symptoms are more severe.

This return of the severe febrile symptoms constitutes what is called the exacerbation, and the period between the time when the fever abates and the development of the exacerbation is called the period of remission. Remissions and exacerbations are the characteristic symptoms of a remittent fever when it is fully developed, at which time a morning

remission is the rule, though the time of the first paroxysm varies. If the exacerbation begins at noon, it will usually decline about midnight, and the remission will last until about noon the next day. In very severe cases there may be a double exacerbation, one at noon, the other at midnight, the remissions being in the evening and morning. The second exacerbation is similar to the primary in its attendant phenomena, except that it is more severe and of longer duration, ends in a less profuse perspiration, and the remission is not so well marked as the first.

On the third day, at about the same hour, or a little earlier, we again have the exacerbation, which has a still longer duration, is of greater severity, and is followed by a more incomplete remission. If the disease goes on from day to day, the remission becomes less and less distinct, and the case becomes dangerous just in proportion as it loses its paroxysmal character. By the end of the first week the remission can no longer be detected, and the fever becomes a continued fever, without any marked daily variation in temperature or pulse. As the remissions become less and less distinct, with each returning exacerbation the tongue becomes more and more parched, sordes collect upon the teeth, the countenance becomes dull and heavy, distress and pain in the epigastrium continues, and is accompanied by tenderness, although the senses of the patient are so dulled that he may scarcely complain of it; the vomiting is not so constant, and is of a less distressing character; constipation, which was probably present at the commencement of the fever, has now given way to diarrhœal discharges, which are usually of a brownish color. With the diarrhœa there is some fulness of the abdomen, and some local tympanitis. The pulse is increased in frequency, and has perhaps reached 120 or 130, is small, thready, and feeble, while at the onset of the disease it was full and compressible. The patient slips down in the bed, picks at the bed-clothes; there is subsultus and difficulty in deglutition, and the tongue is protruded with difficulty, as in the severer forms of typhoid fever. In other words, the patient has passed into a condition closely resembling

that of one who has entered the third week of a typhoid fever, with this exception, there is no eruption.

The diarrhoea, abdominal disturbance and tympanitis, and often the tenderness over the ileo-cæcal region, the typhoid tongue, and the low muttering delirium, closely allies this stage of simple remittent fever to typhoid fever; but the absence of the rose-colored spots and the typical range of temperature of typhoid fever are sufficient to distinguish it from that fever.

After these typhoid symptoms have continued a week or ten days, if the case is to terminate in recovery, remissions recur and become more and more distinct, until finally there is no exacerbation, and the patient passes into a state of convalescence. If, however, a fatal termination is to take place, the remissions will not recur, but the typhoid symptoms will become more marked, and the patient will finally die from exhaustion or from complications. Of all the symptoms which attend simple remittent fever, nausea and vomiting are the most constant and the most distressing. I have seen patients, after the temperature had fallen to its normal standard, suffer for weeks from gastric disturbance, attended by more or less jaundice.

If, in the progress of a remittent fever, the exacerbation occurs a little earlier each day, then treatment is not controlling it, but the disease is gaining ground; the fever is then said to be *anticipating*, and you may be almost certain that the disease is passing from a distinct remittent to a continued remittent.

If, on the other hand, the exacerbation occurs a little later each day, the fever is said to be *postponing*, and you may be sure that you are controlling it, and that, as the remissions become longer, the exacerbations will become shorter and less severe, until the patient reaches complete convalescence. The thermometer will indicate to what extent the disease is being controlled.

This is the history of what may be regarded as simple remittent fever. It begins with a chill, is followed by distinct exacerbations and remissions, and, if not controlled by treatment, becomes a continued fever; then, after a week,

perhaps a longer time, the remissions recur again until convalescence is established, or the typhoid symptoms become more marked, the remissions do not recur, and death ensues.

If a simple remittent fever is protracted, the typhoid symptoms which are developed do not stamp it with a typhoid character; they are such symptoms as are liable to occur in any acute, infectious disease.

BILIOUS REMITTENT FEVER.—In a certain proportion of cases, in all endemics of remittent fever, vomiting of “bilious” material, and *jaundice* are prominent symptoms, the skin often becoming so yellow that the patients present an appearance similar to those suffering from yellow fever; with this yellow discoloration of the skin there is an unusual tenderness on pressure over the hepatic region. Under such circumstances this fever has been named “*bilious remittent*.”

By some of the older writers it has been described as an idiopathic fever, distinct from remittent or any other form of malarial fever. Medical literature, however, contains no facts in support of such a view. The pathology and symptomatology of the fever described by writers under the head of *bilious remittent fever* differ in no respect from those of simple remittent, except that the fever is accompanied by symptoms of more than usual hepatic and gastric disturbance. My own experience leads me to regard it as a form of simple remittent, accompanied by a more than usually severe gastro-hepatic catarrh, and that it is not entitled to a separate place in the nosology of fevers.

INFANTILE REMITTENT FEVER.—In this connection it is perhaps well that I should refer for a moment to a condition which has received the name of infantile remittent fever.

It is a matter of every-day experience that children are subject to certain gastric and intestinal derangements, which are attended by more or less fever, which is very apt to assume a remittent type. Such fevers cannot, however, be regarded as specific diseases, for they are developed independent of any specific fever poison, and are only symptomatic of some local irritation. There is a form of

mild typhoid fever which is often met with in children, especially in the autumn, which has also incorrectly received the name of infantile remittent fever. In this class of cases the usual symptoms of typhoid fever are so modified by age that the fever assumes a remittent type. The presence of rose-colored spots, and the characteristic typhoid lesion of the intestines, will determine the true nature of these fevers.

In malarial districts you will meet with a simple malarial remittent in children, which does not differ from the simple remittent of adults, and does not, therefore, require a separate description. Remittent fever in children is more liable to be followed by malarial cachexia than in the adult.

DIFFERENTIAL DIAGNOSIS.—I have already given you the rules by which you are to distinguish a simple remittent from a simple intermittent fever, and it is not necessary that I should repeat them.

The differential diagnosis between remittent and typhoid fever is often attended with difficulty, if the patient is not seen until the second week of the disease, but if he is seen at the very onset of the fever, it is hardly possible to mistake these two forms of fever the one for the other. The sudden advent of a remittent is in marked contrast to the slow development of a typhoid fever. Besides, they widely differ in the range of temperature during the first week of their development. In remittent there is a distinct remission, and you need not doubt as to the type of fever after the first, certainly not after the second, remission has occurred.

Again, you have the gastric symptoms, which are much more severe in remittent than in typhoid. By these symptoms alone you will be able, in many instances, to make a differential diagnosis. If, however, the fever has been protracted to the third week, and the remissions are slight or altogether absent, although many of the symptoms of typhoid fever are present, the absence of the rose-colored spots is sufficient, taken in connection with previous history of the patient, to establish the diagnosis. Should you be still in doubt, place a drop of the patient's blood under

the microscope, and in nearly every instance, if the case be one of remittent fever, pigment granules will be seen, which at once settles the question, as pigment granules are not found in the blood of typhoid fever patients.

Finally, remittent fever is developed only in malarial districts, and there can be no difficulty in making a differential diagnosis, if the patient resides in a non-malarial district, and is not known to have been exposed to malarial influences.

If hemorrhages occur during the course of a remittent fever, the blood proceeds from the mouth, nose, urinary organs, and bowels; while in the advanced stages of typhoid fever it rarely occurs, except from sloughing of the intestinal glands.

Simple remittent fever may be distinguished from yellow fever by its high range of temperature, by its daily exacerbation and remission, by the presence of pigment in the blood, and in most cases by the absence of albumen in the urine, which is present in yellow fever.

In remittent fever, hemorrhage from the mucous surfaces, especially from the mucous membrane of the stomach, is of rare occurrence, while in yellow fever it is frequently present.

Death often occurs on the third day in yellow fever, but in the severest cases of remittent fever not before the seventh day.

Remittent fever may be confounded with pyæmia and septicæmia, but their differential diagnosis has already been sufficiently considered under the head of intermittent fever. The differential diagnosis between remittent and typho-malarial fever will be considered when I come to the latter disease.

Prognosis.—The prognosis in simple remittent fever is good; death should rarely occur. Even cases of the severe types of this fever should terminate in recovery, if skilfully managed, especially if they are seen in the early stages of the disease.

You must remember that the type of this fever varies very much according to locality. The remittent fever

which we see in this city is of a mild type. In that form which prevails in many parts of the West and South a fatal termination is of frequent occurrence.

There is a type which soon loses its remission, and becomes a pernicious malarial fever, the prognosis of which is unfavorable.

The prognosis will also be modified by the condition of the patient at the time of the attack, and by the character of the epidemic which is prevailing.

I have already indicated the symptoms by which you are to determine whether recovery is to take place, or the case is to terminate fatally. The fact that the exacerbation is delayed or rendered less severe, is a favorable indication, unless the patient becomes more and more overwhelmed by the malarial poisoning, which condition is shown by a high range of temperature and a tendency to coma, or by the patient's passing into a typhoid state. The early subsidence of gastric symptoms, headache and a decrease in the frequency of the pulse, are favorable signs. Distinct remissions, accompanied by moderately free perspiration, indicate an approaching favorable change. On the other hand, if the fever is more continuous than paroxysmal, with a pulse becoming daily more feeble and more frequent, if there is a tendency to collapse at the close of the exacerbations, with signs of extreme exhaustion, danger is indicated.

The average duration of this fever is two weeks.

As this fever varies so greatly in severity at different times and in different localities, it is impossible to determine its average rate of mortality.

LECTURE XIII.

PERNICIOUS FEVER.

*Treatment of Simple Remittent Fever.—Morbid Anatomy.
—Etiology.—Symptoms.*

WE shall have completed the history of simple remittent fever when we have considered its treatment.

When speaking of the treatment of typhoid fever, I stated to you that the fact was constantly to be borne in mind that there was no agent by means of which we could shorten its duration or arrest its development. The contrary is true in this disease, for we have means at our command by which, in the majority of cases, it can be controlled, and by which, in all instances, its duration may be very much shortened. It is hardly necessary for me to speak of such remedial agents as blood-letting, emetics, cathartics, and diaphoretics, which have all been employed in the treatment of this fever, for they have all been supplanted by a *single remedy*. Perhaps there is no more difficult lesson for a young practitioner to learn, when brought to his first case of remittent fever, just as the patient is passing into his first exacerbation, certainly if he has reached his second, than to restrain himself from resorting to a vigorous antiphlogistic plan of treatment. As he feels the burning heat of the skin and the full, bounding pulse, and sees the flushed face and congested eye, and listens to the complaint of intense pain in the head and limbs, of unquenchable thirst, and burning pain in the epigastrium, he is almost impelled to resort to some, or all, of the more vigorous so-called antiphlogistic remedies; but

in this fever it is true, as in the other forms of fever which have been engaging our attention, that these violent symptoms are due to a blood-poison which is exerting its specific effect upon the nerve centres. It is this, not an inflammatory process, that we have to contend with. Experience has proved that this poison cannot be removed from the system by any of the so-called eliminative methods of treatment. If you deplete this class of patients to any extent, you hasten the development of those typhoid symptoms which are especially to be avoided. Persons living in malarial districts are never up to the normal standard of vigor, and, consequently, are in a condition to be affected unfavorably by any plan of treatment or by any remedial agents which shall enfeeble the vital powers.

The first thing to be done in the successful management of this fever is to place your patient under the best possible hygienic surroundings. The same care should be exercised in the arrangement of the sick-room as has already been proposed in the management of typhoid fever. Those who have seen most of remittent fever in its severer forms recommend that the treatment of each case be commenced by administering a mercurial purge. They claim that there is always more or less engorgement of the liver, spleen, and mucous membrane of the stomach and intestines, and that, so long as these organs remain in this condition, no plan of treatment will be successful.

However great may be the differences of opinion in regard to this, all agree that the sulphate of quinine should be used in the treatment of this fever. Practitioners differ, however, as to the mode of its administration, but all are united in its use. Some maintain that it has greater power over the disease when administered in small doses, repeated at short intervals; others maintain that it should be given in one or two large doses during the remission, an hour or two before the commencement of the expected exacerbations. Again, others claim that the quinine has its greatest power over the fever when administered during the activity of the febrile excitement. A few years ago this subject was carefully studied by those engaged in the Eng-

lish Medical Service in India. Under the direction of the Surgeon-General in that department quinine was administered at different periods in the course of the fever. For example, one surgeon gave quinine at the commencement of the exacerbation, another gave it immediately after the exacerbation had passed its height and as the sweating stage was coming on, another gave it immediately preceding the exacerbation, and others gave it during the remission. This plan was adopted in order to determine with positiveness when the smallest amount of quinine would have the greatest controlling effect over the fever. From the various branches of the department reports were made to the Surgeon-General, and from these reports the conclusion was arrived at, that quinine, administered during the time of the exacerbation, had not only a greater influence in diminishing the severity of the disease, but it also more completely controlled the fever, and more markedly shortened its duration than when it was administered during the remission. From the conclusion arrived at from their reports, and from my own experience, I should not hesitate to administer quinine at any time during the period of exacerbation or remission. My rule is to give ten or twenty grains at a dose, according to the severity of the fever, and repeat it every two hours until cinchonism is produced. When cinchonism is reached, although the fever may not be controlled, it is well to stop its administration until twenty-four hours have elapsed; by doing this you will be better able to determine the antiperiodic power of the drug. If you find that the exacerbations do not disappear, but are delayed and are less severe, you may be sure that you are controlling the fever. If, notwithstanding this free use of quinine, the exacerbations are more severe and longer in duration, and the remissions less frequent, and typhoid symptoms are manifesting themselves, stimulants may be demanded. Even large doses of stimulants may be required to sustain the patient while he is passing through this period of the disease.

Remittent fever is not, like typhoid fever, a disease of days or weeks. In its severer forms, no time should be

lost while waiting for the action of cathartics or other remedial agents which are supposed to be of importance, but you should at once commence the administration of quinine. When the disease has reached its second or third week, and there is no evidence that the patient is passing on towards recovery, you must commence a second time the administration of large doses of quinine; in this way you may arrest the progress of the fever. If, after a second cinchonism is produced, the fever is not arrested, you must again omit for a few days the administration of quinine; then repeat the large doses a third time. It is much better to proceed in this way with the remedy than to keep your patient in a continued state of cinchonism. It is not necessary to enumerate the long list of drugs which at different times have been proposed as specifics in this fever, all of which, by common consent, are now regarded as far less reliable than quinine. The important thing is to know how and when to administer quinine.

There are certain palliative measures which it is sometimes important to employ. If the exacerbations are very intense, the headache very severe, and the restlessness or other febrile symptoms are not relieved by full doses of quinine, you may resort to the use of cold for its antipyretic effect, the same as in typhoid fever.

Frequently, in mild cases, sponging the surface with tepid water is not only grateful to the patient, but it has a controlling influence over the fever. If vomiting is constant, severe, and exhausting, hypodermics of morphine will be found of service.

As in typhoid, the treatment of this fever is expectant, save in the use of quinine.

PERNICIOUS MALARIAL FEVER.

I now pass to the next in my list of malarial fevers, which I shall describe under the term of pernicious fever. This form of fever has received other names, at different times and in different localities. It has been called *congestive*

fever, ardent fever, tropical typhoid fever, and pernicious fever.

I have adopted the latter name, for it seems to me to be not only the most appropriate, but the one which at the present time is most generally adopted. It is true that in the majority of cases there is more or less congestion of the internal organs, and sometimes the patient is overwhelmed by these congestions, but in a large number of cases no such congestions exist, and under such circumstances the designation pernicious is mostly to be preferred.

It is the most severe and dangerous form of malarial fever. It may be intermittent or remittent in character, and may assume any of the types of periodical fever, but the quotidian and tertian types are the most common. Sometimes its pernicious character is clearly marked at the onset of the fever, during the first paroxysm; at other times it comes on insidiously, and its pernicious character is not suspected until after the occurrence of two or three paroxysms.

There are several well-marked and distinct varieties of pernicious fever—the most common and most important of which are the *comatose*, the *delirious*, the *algid*, and the *gastro-enteric*. Almost every locality where pernicious fever prevails gives to the fever some distinctive peculiarity.

Pernicious fever not infrequently appears as an epidemic, although sporadic cases are met with in those regions where simple intermittent and remittent fevers prevail. I have seen six well-marked cases of pernicious fever in this city during the past year.

MORBID ANATOMY.—The anatomical lesions of pernicious fever are similar in kind to those found in simple intermittent and remittent fevers, but they differ very much in degree. For instance, you will find similar blood-changes, the most striking of which is the presence of free pigment in the blood. But the pigmentation is more abundant, and the pigment material may be in the form of granules, or in the form of plates, or it may even have a cellular outline. The abundance of the pigment, and the extent of the pig-

mentation will vary according to the severity of the fever. But in all cases there is some free pigment in the blood. This pigment is not often present in the blood in simple intermittent, unless the fever has been prolonged, and in simple remittent it is never as abundant as in pernicious fever.

The other changes in the different organs and tissues of the body are very similar in character to those to which I have already referred in connection with the morbid anatomy of intermittent and remittent fever.

As the varieties in type of this fever are as numerous as the localities in which they occur, and as the type in any locality may change with every succeeding year—that is, the type of one year may be very unlike that of the preceding or following year—you see that it is very difficult even to classify its different forms.

The slight variations which are met with in the pathological lesions of the different varieties, are still more difficult of description and classification. For instance, there is one variety which is characterized by a tendency to coma, called the *comatose variety*; another is characterized by a tendency to a peculiar form of delirium, termed the *delirious variety*; still another which is characterized by a marble-like coldness of the surface, called the *algid variety*; again, we have one which is characterized by vomiting and purging, or choleraic symptoms, termed the *gastro-enteric variety*; then one in which there is acute jaundice, termed the *icteric variety*; then one in which there are profuse hemorrhages, termed the *hemorrhagic variety*, and still another in which there is profuse diaphoresis, termed the *colliquative variety*.

These are the more common varieties of pernicious fever. There are still others of such rare occurrence that it is hardly necessary that I should mention them, as they are slight variations due to local causes. None of these are distinct fevers, but different types of the same fever.

As in scarlatina, measles, and small-pox, we have different names assigned to different types of the same disease, so all these forms of pernicious fever are simply different

manifestations of one and the same fever, due to one and the same cause, namely, malarial poisoning.

You will find the post-mortem appearances in pernicious fever varying with the intensity of the malarial infection, and the peculiar atmospheric conditions under which the fever is developed.

In some instances there will be evidences of intense engorgement of the blood-vessels of the brain, and the entire brain substance will be more or less thoroughly stained with pigment material. In others, minute blood-extravasations will be found scattered here and there throughout the substance of organs. Small blood-extravasations into the spinal cord, accompanied by more or less pigmentation, is very apt during life to be attended by tetanic spasms. In persons dying of pernicious fever after the third attack, I have found all the organs of the body pigmented.

Sometimes you will find intense engorgement of the liver, that is, the most marked post-mortem changes will be found in that organ, and the amount of pigmentation present will correspond with the intensity of the congestion. With intense engorgement of the organ there are usually blood-extravasations.

Occasionally, infarctions occupy the spleen, around which there will be a mass of pulpy material. The spleen is more frequently found softened in this form of malarial fever than in those forms already described. Although enlarged, it is usually softened and of a darker color than normal. It is sometimes so soft that it closely resembles the spleen of typhoid fever, and is merely a pulpy bloody mass, though in size it is larger than in typhoid fever. If not softened it may have infarctions scattered through its substance. Marked pigmentation of the tissues of the body, corresponding in amount with the peculiar symptoms present during life, a tendency to enlargement and softening of the spleen, enlargement of the liver with deeper pigmentation than is seen in any other organ of the body, are among the more common pathological lesions of this form of fever. It is unnecessary to describe in detail that enlargement of the capillary vessels which occurs as a necessary result of this

intense engorgement. Sometimes the kidneys and the lungs are the seat of this intense hyperæmia, as the result of which the functions of these organs are more or less extensively interfered with.

ETIOLOGY.—The exciting and predisposing causes of pernicious fever differ from those of the simpler forms of malarial fever only in degree, not in kind, but a higher range of temperature is requisite for the development of pernicious fever. It prevails only in those localities where the average range of temperature, for a time, reaches 65° F.

SYMPTOMS.—Pernicious fever may commence abruptly, but generally the premonitory symptoms which mark its development do not differ from those which mark the development of simple intermittent and remittent fever. In most varieties the attack commences with a chill, which is unusually severe and prolonged. In many cases you will have a distinct malarial paroxysm of either the intermittent or remittent type, and the pernicious character of the fever is engrafted upon it. In other words, you may have the attack commencing with a distinct intermittent fever paroxysm of the quotidian type, but rarely more than two of these intermittent paroxysms will occur before it assumes the pernicious type, if it is to become a pernicious fever; or you may have a remittent fever with a distinct exacerbation and remission, which may go on for four or five days before its pernicious character will be developed.

The milder form either gradually passes from a simple intermittent into a pernicious fever by a progressive increase in the severity of the symptoms, or a single paroxysm of not unusual severity is suddenly followed by a pernicious one; this latter seldom proves fatal, unless it has been repeated for the second or third time. Again, you may have a distinct chill followed by a condition that you will at once recognize as one of the varieties of pernicious fever. The ushering-in symptoms will always vary with the type of disease which is about to be developed.

I shall not attempt to describe the phenomena that attend all these different varieties, but will only speak of those

most commonly met with, and detail their prominent and leading symptoms.

COMATOSE VARIETY.—We will suppose that a patient has a distinct paroxysm of one of the simpler forms of malarial fever, either intermittent or remittent, with no special phenomena attending it, except that he has had a more severe headache than ordinarily occurs in a simple form of malarial fever; with this perhaps there has been vertigo, a stammering and indistinctness in the speech, an inability to talk with freedom, and a more than usual tremulousness during the hot stage. From this condition he passes as usual into the hot stage of an intermittent, or rapidly into an exacerbation of remittent, then into a state of stupor and unconsciousness, and finally lies upon his back, with a flushed face, congested conjunctiva, dilated pupils; slow, deep, stertorous respiration, and perhaps a very slow pulse, or, if slow at first, it may soon become frequent. The axillary temperature ranges from 105° F. to 107° F. The patient is now nearly unconscious; he is apparently paralyzed; the urine is retained in the bladder, and the bowels move involuntarily. If the pulse is slow, it is full and hard. The respiration becomes more and more stertorous, unconsciousness becomes more and more complete, until he finally dies in a state of complete coma. Usually, however, a moisture makes its appearance within twelve hours from the commencement of the first paroxysm, and the patient awakes to consciousness in the midst of a profuse perspiration. The headache and giddiness have now nearly passed off, and if the fever which preceded it was remittent, there may be a well-marked remission; if it was an intermittent, there may be a distinct intermission. With the next remittent exacerbation or the hot stage of intermittent, the pain in the head, giddiness, unconsciousness, and all the symptoms already described will return more intense in character than before, with the coma and stupor, and perhaps with the second attack the patient passes into a fatal coma. These are the leading phenomena which attend the most common form of pernicious fever.

In this variety patients sometimes pass into a condition

of apparent death, which may last for hours. Some are perfectly conscious, seeing and hearing everything which occurs around them, while they are unable to move or utter a sound; others pass into a state of unconsciousness, but the respiratory movements and the heart's action are not perceptible. Even though the strongest counter-irritants be applied to the surface, there is no sign of life, until, at the beginning of the sweating stage, the patient comes to himself.

If a patient survives the first paroxysm of this form of pernicious fever, quite probably he will die during the second. With each successive paroxysm the prognosis becomes more and more unfavorable; patients sometimes lie in a comatose condition for days, and finally die apparently from cerebral compression.

DELIRIOUS VARIETY.—In this variety your patient, after passing into the hot stage of an intermittent or into the exacerbation of a remittent, becomes delirious. Mild delirium is not uncommon during the progress of an intermittent or a remittent fever, but the delirium now referred to is of a more active character. If then delirium is developed during the exacerbation of a remittent or during the hot stage of an intermittent, which has been preceded by severe headache, dizziness, ringing in the ears, and great restlessness, you may be quite certain that you have to deal with a case of pernicious remittent or of pernicious intermittent fever, especially if pernicious fever is prevailing in the locality. In this variety of pernicious fever there will also be more or less headache during the interval, and perhaps other peculiar cerebral phenomena. The delirium which appears is always violent in character; perhaps the patient will require restraint; he may be disposed to jump out of a window, or in some way to do injury to himself or those around him.

During the paroxysm of delirium the patient's face becomes flushed, his eyes brilliant, the conjunctivæ injected, the pupils dilated, and he is constantly crying, singing, and trying to escape. In those who are extremely anæmic the countenance assumes a pale, sunken aspect. The pulse is full and hard, and the carotids beat violently, the tempera-

ture often reaches 107° F. or 108° F. The patient may remain in this delirious state for hours. Somewhat suddenly he passes from it into a condition of collapse, or he gradually sinks into a coma from which he never awakens. During the whole period the axillary temperature rarely falls below 105° F. In favorable cases the delirium gradually becomes milder, a profuse perspiration comes on, and the patient falls into a prolonged sleep, from which he awakes conscious, though weak and exhausted, with headache and vertigo, but without the slightest recollection of what has passed. These attacks of delirium may be repeated three or four times before a fatal termination is reached, but so much danger attends them, that a second attack should never be allowed to occur if it can be prevented.

In this variety of pernicious fever, other nervous phenomena may accompany or take the place of the delirium, such as epileptiform convulsions, tetanic spasms, etc. The tetanic spasms sometimes assume the phenomena of hydrophobia.

That form of tetanus which occurs in various malarial districts, which is sometimes called sporadic tetanus, I believe will be found to be of this type, and simply a form of pernicious fever.

GASTRO-ENTERIC VARIETY.—In this variety the patient, after he has passed into the hot stage of an intermittent or the exacerbation of a remittent, is seized with almost incessant vomiting and purging. The vomiting and purging is of a peculiar character, altogether unlike that which is sometimes present in the simpler forms of malarial fever. There is blood-stained material, both in the matter vomited and in that discharged from the bowels. In some instances, the discharges may be so reddened as to look like beef-brine or the washings of raw beef; sometimes the proportion of blood is so great as to cause the discharges to have the appearance of clear blood. In some endemics the discharges assume the appearance of rice-water, having no odor, and similar in appearance to those in Asiatic cholera. The patient has no abdominal pain or tenderness, but has a sense of weight and burning in the stomach, accom-

panied with cramp in the calves of the legs, coldness and blueness of the surface, with a small, almost imperceptible pulse, sunken eyes, and the facies of cholera. So closely do these patients resemble in appearance those with Asiatic cholera, that this disease has frequently been mistaken for cholera. During the attack the thirst is most intense. The respiration is peculiar; it consists of a double inspiration, followed by a double sighing expiration. The restlessness is very great, the patient is constantly tossing from one side to the other; sometimes he suddenly, an hour or two before death, springs up and walks across the room. The usual length of the fatal paroxysm is from three to six hours. Patients die in a state of collapse; after the vomiting and diarrhœa have assumed the characteristic appearances already described very few patients recover. As death approaches, the pulse becomes more and more frequent, feeble, irregular, and fluttering in character. The respiration is more and more prolonged and sighing, the skin cold and shrivelled, and covered with a cold, clammy perspiration. It frequently happens when all these symptoms are present that the patient cannot be convinced that he is seriously ill, and wishes to get out of bed and go out of doors.

It is important to remember that these three varieties of pernicious fever, which I have just briefly described, are not always distinct, but the symptoms of one may be mingled with those of another; such mixed cases are very difficult to classify.

LECTURE XIV.

PERNICIOUS FEVER.

Symptoms (continued).—Differential Diagnosis.—Prognosis.—Treatment.

At my last lecture I spoke of the *comatose, delirious*, and the *gastro-enteric variety* of pernicious fever. I now invite your attention to the consideration of another variety, which bears a striking resemblance to the one we last considered. It is termed the *algid variety* of pernicious fever.

Algid Variety.—This variety is characterized by coldness of the surface of the body, while the rectal temperature may range from 104° F. to 107° F. The attack begins with a chill of not unusual severity or duration, but soon after the patient enters the hot stage of the paroxysm; or, during the exacerbation of a remittent, the surface of the body begins to grow cold, while at the same time he complains of a sensation of burning and intense thirst. A cold perspiration soon covers the surface. The pulse becomes slower and slower, falters, and disappears at the wrist. Alternately the extremities and face become cold; only the abdomen retains its normal temperature. The surface has a cold, marble-like feel, but the temperature in the axilla never or rarely falls below the normal standard. In the *comatose* and *delirious* varieties the temperature rises higher than normal, and may reach 106° F. or 107° F., but in this variety it sometimes falls two or three degrees lower than normal. The tongue becomes white, moist, and cold; the breath is cold, and the voice feeble and sunken. The action

of the heart is feeble, often perceptible only on auscultation. The mouth is clean, and the patient seems to himself to be in a comfortable condition, except that he feels exhausted. The expression of countenance is that of death. In its progress this variety of pernicious fever is very insidious. If you are not familiar with it you will quite probably mistake the calm which follows the febrile excitement for relief, perhaps attribute it to some plan of treatment which you have pursued, or to some remedial agent which you have employed. If a patient in one of these paroxysms is to pass on to recovery, the pulse gradually returns in the wrist, and the surface regains its normal feel and temperature. As the warmth returns to the surface the patient passes on to convalescence in the same manner as patients recover from a comatose or delirious paroxysm.

An *algid* pernicious paroxysm is rarely preceded by a distinct intermission, and it rarely has any appreciable remission. Once established, it marches steadily on to a fatal issue, unless arrested by treatment.

There is another variety which you will occasionally meet with, in which a profuse perspiration, called a "*colliquative sweat*," comes on at the end of the fever stage and continues through the succeeding intermission, accompanied by great prostration, feeble heart action, and labored respiration. Upon the second or third return of this sweat the patient sinks and dies apparently from exhaustion.

Again, severe hemorrhage from the stomach, bowels, or kidneys may occur during the sweating stage of a pernicious paroxysm and endanger the life of the patient from sudden syncope.

A mild form of haematuria sometimes occurs independent of a pernicious paroxysm in chronic malarial poisoning.

There is still another variety, concerning which I will say a few words. It is always endemic, confined to certain localities, occurring in those localities when any form of pernicious fever prevails. It is called the icteric variety.

ICTERIC VARIETY.—This variety begins with a violent, long-continued chill, during which jaundice shows itself. The jaundice gradually deepens, and extends over the whole

body. Intense nausea accompanies its development, with a copious vomiting of bile, and a bilious diarrhoea. The patient suffers with a most intense headache, pain in the region of the spleen and over the kidneys, and a feeling of numbness in the limbs. The pulse is small, frequent, and hard. The urine is deep-colored. As the hot stage comes on the pulse becomes more frequent and fuller, the respiration is labored, the skin very hot, the temperature reaching 106° F. or 107° F., and the thirst is most intense. This stage lasts three or four hours, and often terminates in death. If the patient passes into the sweating stage, recovery usually takes place. During the intermission the mind is clear, but the jaundice continues. Unless the disease is controlled by treatment, each succeeding paroxysm becomes more and more severe. This variety is incorrectly called pernicious bilious remittent fever.

If the attack is mild, there is only a slight staining of the skin, but in that form in which there is an apparent arrest of the functions of the liver, the patient may die deeply jaundiced, within two or three days after the first discoloration of the skin has appeared. There is a mild form of so-called bilious remittent fever, to which I have already referred, in which the febrile movement is constant; this is very different from that form to which I now allude, and is better classed under the head of simple remittent.

These different varieties of pernicious fever, of which I have made mention, are almost the only ones ordinarily met with in practice. It seems to me that very properly they may all be considered under the general head of pernicious fever.

Remember that all these different varieties depend on the same blood-poisoning, differing in its manifestations according to the intensity of the poison and the predisposing atmospheric or septic conditions which may exist in the localities where they are developed.

DIFFERENTIAL DIAGNOSIS.—The diagnosis of pernicious fever is sometimes very difficult. In determining whether a given case is, or is not, one of pernicious fever, the first inquiry will be in regard to the character of the prevailing

fever. If pernicious fever is prevailing in the locality, doubtless your diagnosis will be easily made; if, however, the first case in the locality falls under your observation, probably, you will find great difficulty in making a diagnosis, and this difficulty, to a certain extent, will vary with the type of the fever. If, for example, your case belongs to that class in which there is a tendency to comas, delirium, etc., you may confound it with some form of cerebral disease. This form of pernicious fever has been mistaken for *cerebral apoplexy*, *meningitis*, and *acute uræmia*. As a rule, it is not difficult to draw the line between apoplexy and pernicious fever of the comatose or delirious variety.

The constant and prominent symptom of apoplexy is hemiplegia, which is of rare occurrence in pernicious fever. It may occur, but if it does, it is developed slowly. Neither coma nor hemiplegia is ever reached suddenly in pernicious fever. There is a rise in temperature, rapid pulse, and all the phenomena of intense febrile excitement are present before the occurrence of either. On the other hand, in apoplexy the hemiplegia is of sudden development, attended by a slow pulse, irregular, contracted pupils; or, perhaps, one pupil is dilated and the other contracted, and its occurrence is preceded by a sudden loss of consciousness, and not attended or preceded by high febrile excitement. These marked differences between the two diseases will lead you to a correct diagnosis.

As regards mistaking pernicious fever for meningitis, it would seem hardly possible for one familiar with both diseases to make such a mistake in diagnosis.

Though in both diseases the patient reaches a condition of coma, in meningitis days elapse before the coma is reached, and during those days there has been pain in the head, photophobia, delirium extending over a considerable period of time, and then the coma; whereas, in pernicious fever, within twelve hours the patient reaches his condition of coma. Besides, in pernicious fever there will be a history, not only of the prevailing type of malarial disease, which will indicate its character, but there will precede the attack of coma or delirium a distinct malarial paroxysm—

perhaps two of these paroxysms; then the patient will pass rapidly into a state of coma. In these two diseases the condition of the pupil varies. In meningitis, when the patient reaches complete coma, the pupil will be dilated, while in the comatose variety of pernicious fever the pupil may be contracted, dilated, or normal.

The gastro-enteric and cold or algid variety of pernicious fever closely resembles cholera. It may be distinguished from it by the character of the primary discharges. You may reach a time in pernicious fever when the discharges will very closely resemble those of cholera; but they have been preceded by one or two bloody discharges. Then in cholera you will have albumen in the urine, the occurrence of which is comparatively rare in pernicious fever. Then in cholera there are the peculiar surroundings of the patient, the prevalence of cholera in the locality, etc. Yet, in a recent endemic of choleraic-pernicious fever which prevailed along the banks of the lower Mississippi, many prominent physicians maintained that it was an epidemic of Asiatic cholera. When the endemic is at its height it is almost impossible to make a differential diagnosis between the two diseases from the clinical history of the cases; but, when you take the early history of the endemic, at which time the cases at their commencement were marked by distinct intermittent or remittent paroxysms, then the true character of the disease is very readily determined. If in any given case there is still a question whether it is or is not one of pernicious fever, this can be determined with positiveness by placing some of the patient's blood under the microscope, when, if the case be one of pernicious fever, the blood will be found to contain pigment.

The icteric variety of pernicious fever, which often, in many of its phenomena, so closely resembles yellow fever, may be distinguished from it not only by the history of its development, but by the fact that when it prevails as an endemic, those are seized with the fever who have been longest under the influence of malarial poison, whereas new-comers are not usually attacked; in yellow fever districts new-comers are almost certain to contract the disease.

Then the jaundice of yellow fever appears late in the disease, while the jaundice of this form of pernicious fever comes on early, even before the chill passes away. Again, bloody urine is frequently present in this type of pernicious fever, while in yellow fever hæmaturia rarely occurs without the accompanying evidences of nephritic inflammation.

It is hardly necessary for me, under the head of differential diagnosis, to speak of all the different varieties of pernicious fever, for there is one thing—the presence of free pigment in the blood—which settles the question of diagnosis in difficult cases; this is present in nearly every severe case in any form of pernicious fever.

Whenever any of these types of pernicious fever prevail in the region where you are located, you will soon become familiar with their peculiar phenomena, and thus be able to make an early diagnosis. You must bear in mind that, though you have become familiar with one variety of this fever, you are by no means prepared to make an early diagnosis of any other variety, for the algid and comatose varieties differ as widely in the phenomena which attend their development as though they were distinct diseases and did not depend upon the same poison.

PROGNOSIS.—In all varieties of pernicious fever the prognosis is unfavorable. Unless you are able to control the disease before the occurrence of the second paroxysm, usually the case will terminate fatally. In all cases the prognosis will depend, to a great degree, upon the character of the prevailing endemic or epidemic, as also upon the stage of the epidemic, for the ratio of mortality is always greater during the early period of an epidemic than during its decline. During the latter part of an epidemic you may think you are managing your cases better because fewer patients die, while the good results are due to the fact that the epidemic is on the decline. All observers agree that the prognosis is better in every variety of pernicious fever if there are distinct intermissions, however short may be their duration. If the paroxysm does not last more than twelve hours, and terminates in a distinct remission, the prognosis is far better than when one parox-

ysm follows another without any distinct remission. If the paroxysms are increasing in severity and duration, the patient is liable to die in the third or fourth paroxysm.

Unquestionably the most favorable cases are those of the tertian type. Those varieties in which the cases most frequently terminate fatally are the gastro-enteric and the algid; those in which the cases are most likely to recover are the comatose and delirious.

In every case the prognosis is very much influenced by the age and condition of the patient, and by the presence or absence of complications. The mortality is greatest among the very young and very old, and among the intemperate.

Patients with pernicious fever may die suddenly during a paroxysm, or the paroxysms may be prolonged and run into each other, and the patient may finally pass into a typhoid or collapsed condition.

In every variety of pernicious fever you may be aided in making a prognosis by remembering what I am about to state.

If the second or third paroxysm is not attended by signs of intense visceral congestion, if it declines with profuse warm sweats, if it has been preceded by distinct intervals, if the urine is free and the appetite early returns, you may safely prognosticate a speedy recovery. On the other hand, if the second or third paroxysm is protracted and accompanied by great anxiety and restlessness, with active delirium and a tendency to coma, with coldness of the surface; if there is intense pain in the epigastrium, with tingling of the surface, and scanty and high-colored urine; if there is profuse vomiting and purging, bleeding at the nose and cold, colliquative sweats; if the pulse becomes small and feeble, or the radial pulse is imperceptible, the danger is very great, and a fatal issue is almost certain, either immediately or in the fourth or fifth paroxysm. Sometimes severe and fatal dysentery comes on at the end of a paroxysm; at other times, as the paroxysm subsides, the fever assumes a typhoid type, and, after a period of continued fever ranging from ten to twelve days, it terminates fatally.

TREATMENT.—The expectant plan of treatment, which has

been proposed for the management of some of the forms of fever which have engaged our attention, cannot be practised in the treatment of pernicious fevers. The alarming symptoms crowd upon one another with great rapidity, and it is only by prompt and vigorous measures that in the severe forms of the disease you will be able to rescue your patient from impending death. The issue of life or death often hangs upon a single hour.

Some have proposed, before administering the only specific which we possess capable of controlling this disease, to produce free purgation by the administration of cathartics; others to bleed and freely vomit the patients. If the case is one of the gastro-enteric variety, emetics and purgatives are certainly very plainly contra-indicated. It is now a well established fact that in all varieties of pernicious fever patients do not bear depletion. In India, where the most severe forms of pernicious fever prevail, the English surgeons are very positive in their testimony upon this point. All forms of depletion have been abandoned in the India service.

Although stimulating enemas and friction to the surface may act as aids in the management of the algid and delirious varieties, they must not be relied upon for any controlling influence which they may have over the disease.

Those who have had the most extended opportunities for testing the different remedies and plans of treatment which have been employed in the management of this fever, are united in the opinion that quinine and opium are the only agents which can be relied upon for controlling every variety.

In the treatment of this fever my own experience is not extended; consequently, I am compelled to give you the teachings of those who have written upon this fever.

So far as I have been able to arrive at conclusions from my readings, as well as from my limited experience in the treatment of this disease, I am convinced that in the majority of cases, by the use of opium and quinine hypodermically, we may hope to control it, and thus save the life of our patient.

In fact, the hypodermic use of these drugs has inaugurated a new era in its treatment, for in a large proportion of the severer forms it is impossible to get the full effect of either of these remedies by the ordinary methods of their administration, the usual avenues for their introduction into the system being closed.

The solution of quinine, commonly employed by the English surgeons for this purpose, is made by adding one hundred and fifty grains of quinine and fifty drops of dilute hydrochloric acid to four ounces of water, and then evaporating the solution to two ounces. Of this, thirty drops may be administered at each injection. Some add carbolic acid to a solution of quinine in dilute sulphuric acid; the carbolic acid is added to prevent abscess at the point where the injection is introduced.

The formula for this solution is as follows :

R. Quinia disulphatis..... grs. l.
 Acid sulphurici..... ℥. v.
 Acid carbolici..... ℥. ij.
 Aquæ destillat $\frac{3}{4}$ i.
 M.

Thirty minims is the quantity usually administered at each hypodermic injection; it represents between three and four grains of quinine. I have recently used the following:

R. Quinia sulph..... $\frac{3}{4}$ i.
 Hydrobromic acid ... 3 ij.
 Aquæ destillat 3 vi.
 M.

Thirty minims contain four grains of quinine.

Whatever solution you may use, administer from five to seven grains of quinine every hour until the paroxysm has passed, then continue its use in three grain doses every four hours.

With the quinine of the first hypodermic injection administer one-fourth of a grain of morphia. The morphia should be administered with each dose of quinine until the patient

is brought fully under its influence, without regard to the stage of the paroxysm.

During the past few years a remedy known as "Warburg's Tincture" has been quite extensively employed in the treatment of pernicious fever by the India surgeons. When this remedy was first employed, its ingredients were unknown, and on this account it was not generally made use of by the profession. All those who used it claimed that it more successfully controlled the fever than opium and quinine, or any other remedy that had hitherto been employed. The results claimed for it were really astonishing.

Recently, the formula for making this tincture has been published in the London *Lancet*. I will give it as published.

FORMULA.

Warburg's Tincture.

℞. Aloes (Socotr.) librom,
Rad. rhei (East India),
Sem. Angelicæ,
Confect. Damocratis, ana uncias quatuor,
Rad. Helenis (s. Enulæ),
Crocī sativi,
Sem. Fœniculi,
Cret. præparat, ana unc. duas.
Rad. Gentianæ,
Rad. Zedoariæ,
Pip. Cubeb.,
Myrrh. Elect.,
Camphoræ,
Bolete laricis, ana unciam.

The above ingredients to be digested with 500 ounces of proof spirit in a water-bath for twelve hours; then expressed and ten ounces of disulphate of quinia added, the mixture to be replaced in the water-bath until all the quinia be dissolved. The liquor, when cool, is to be filtered, and is then fit for use.

It will be seen that each half-ounce of the tincture contains seven and a half grains of quinine. It is recommended to give half an ounce of this tincture at the onset of the paroxysm; if this does not control it, the same quantity must be repeated in four hours. If it cannot be retained by the stomach, it may be administered by the rectum, in ounce doses every hour. It is claimed that the tincture is retained by the stomach when all other remedies are rejected. Prof. Maclean says that he has seen the most hopeless cases—those manifesting a degree of severity which seemed to preclude the possibility of recovery—commence to convalesce as soon as the patient was brought under the influence of this remedy. I will quote Prof. Maclean's rules for its administration :

“The tincture is administered in the following manner : One-half ounce (half of a bottle) is given alone, without dilution, after the bowels have been evacuated by any convenient purgative, all drink being withheld ; in three hours the other half of the bottle is administered in the same way. Soon afterwards, particularly in hot climates, profuse, but seldom exhausting, perspiration is produced ; this has a strong aromatic odor, which I have often detected about the patient and his room on the following day. With this there is a rapid decline of temperature, immediate abatement of frontal headache—in a word, complete defervescence, and it seldom happens that a second bottle is required. If so, the dose may be repeated as above. In very adynamic cases, if the sweating threatens to prove exhausting, nourishment in the shape of beef-tea, with the addition of Liebig's extract and some wine or brandy of good quality, may be required.”

No special rules can be laid down in regard to the administration of stimulants in pernicious fever ; the condition of the patient must be your guide. They are simply means used to aid in carrying a patient over a dangerous period. Their continued use in large quantities is objected to by those who have had the most extended experience in the management of this fever.

I will repeat in as few words as possible the important

things to be remembered in the treatment of pernicious fever. Do not wait for the action of a calomel purge. Do not resort to any depleting measures ; patients with this fever cannot bear depletion. However mild the paroxysm may be, no time should be lost ; bring the patient as rapidly as possible under the influence of quinine and opium, or, if " Warburg's Tincture " is used, administer it in full doses as early as possible, and continue its administration until convalescence is fully established.

LECTURE XV.

DENGUE FEVER.

Morbid Anatomy.—Etiology.—Symptoms.—Differential Diagnosis.—Treatment.—Chronic Malarial Infection.

BEFORE leaving the class of fevers which has just been engaging our attention, I wish to say a few words concerning a fever which, although it may not properly be included in the list of malarial fevers, yet it seems to me that it can be best considered in this connection. It has received the names, *dengue*, *break-bone*, and *dandy* fever. It is neither an intermittent nor a remittent fever; nor is it a pernicious fever. It is an acute disease which appears as an epidemic in hot climates. It is characterized by a febrile excitement remitting in its character, and is accompanied by more or less intense arthritic pains, attended by the development of a papillary eruption resembling that of measles.

MORBID ANATOMY.—The morbid anatomy of this variety of fever does not differ essentially from that of the severer types of malarial fever, except that a cutaneous eruption commences on the palms of the hands and extends rapidly over the entire body. In most cases, arthritic changes of a rheumatic character are present; usually the external lymphatic glands are somewhat enlarged.

This disease seems to be an exanthematous malarial fever, with a rheumatic or neuralgic element.

ETIOLOGY.—Dengue or break-bone fever may prevail epidemically in well marked malarial districts, or it may be

met with as a sporadic disease. Its infection has been carried in clothing from one seaport to another. Some claim that the disease depends upon a specific contagion; but its contagious character has not been established.

The intensity of the malarial poison unquestionably has some influence in increasing or lessening the severity of this fever. In districts slightly malarial usually its type is mild; but in districts strongly malarial its type is severe. It attacks all classes and all ages, rich and poor, black and white, the very young and the very old. Occasionally it has occurred as the precursor of yellow fever. In 1827 a very extended epidemic of this fever prevailed in the West Indies; during the prevalence of this epidemic, the specific poison of the disease was transported in clothing and merchandise to many neighboring seaports.

SYMPTOMS.—The period of incubation is estimated from three to five days. The initiatory symptoms are very sudden in their manifestation, and the development of the fever is very rapid. In the majority of cases, the earliest symptoms are headache, photophobia, great restlessness, chilliness alternating with flashes of heat, and pain in the back, limbs, and joints; the small joints swell, and there is soreness and stiffness of the muscles. The skin becomes hot and dry, and in some instances the temperature reaches 107° F. The pulse is rapid, ranging from 120 to 140 beats per minute. The face is flushed and the eyes red and watery. After the fever has continued about twelve hours, the pains in the joints become intense, the pain in the back shoots down the sciatic nerve, and now nausea, vomiting, and pain in the epigastrium are usually the prominent symptoms.

At this stage of the fever the lymphatic glands become involved; the inguinal glands are first affected, then those in the axilla and neck; they increase very rapidly in size, and become exceedingly tender. The testicles enlarge, or rather the epididymis, and the swelling continues until the subsidence of the other symptoms. The active febrile excitement continues from twelve hours to three or four days, when it subsides, leaving the patient in an exceed-

ingly feeble and prostrate condition. Sometimes the fever abates suddenly, with the occurrence of critical symptoms as in relapsing fever, such as profuse sweats, diarrhoea, or epistaxis. Then the patient is in a passive condition for two or three days, and passes into the period of remission. The pains now become less, the glandular swellings diminish, there is less of febrile excitement, but it does not entirely subside. After two or three days a second paroxysm occurs, and the fever returns. About the same time intervenes between the first and second paroxysm as occurs between the first and second paroxysm of relapsing fever. When the fever returns it is more intense, the pain in the joints is more severe, and finally, when the fever has reached its height and the pain is most intense, usually on the fifth or sixth day, an eruption makes its appearance. It first appears upon the palms of the hands, then upon the neck; soon it extends downward and is seen upon the chest, and finally spreads over the entire body. Usually it is papillary in character and very closely resembles the eruption of scarlatina. In most cases, as soon as the eruption is developed, the febrile symptoms subside and the patient goes on to convalescence.

From the intense arthritic pains accompanying the papillary eruption, and from the glandular swellings, you will be able to recognize this peculiar type of fever. As the second paroxysm of fever subsides, the patient is left with stiffness and soreness of the joints, which sometimes does not pass away for weeks. Occasionally the disease assumes a typhoid type, the tongue becomes coated with a dark brown coating, the gums become red and spongy, the pulse is slow and feeble, and the surface is covered with a cold sweat. As soon as the eruption appears, the patient is generally free from fever, and passes on to a rapid and complete convalescence.

In very severe cases the pain in the testicles will continue after the subsidence of the fever, and a serous effusion will take place into the tunica vaginalis. The joints will remain painful and flabby. There will be extreme nervousness and anxiety. The heart's action will be intermittent,

and the lymphatic glands, which have been enlarged, form indurated tumors; they very rarely suppurate. The duration of this fever varies with the period of remission. Its average duration is about eight days.

In those epidemics where there is an absence of articular pains, the mucous membrane of the mouth and throat becomes involved.

The course of the disease may be divided into periods. First, that of febrile exacerbation, lasting two or three days, then an intermission of two or three days, then a second febrile exacerbation which lasts two or three days, then convalescence begins.

DIFFERENTIAL DIAGNOSIS.—This fever may be confounded with rheumatism, or with remittent fever. In some of its phenomena it closely resembles relapsing fever.

It may be distinguished from remittent fever by the persistency of the rheumatic and neuralgic pains, by the cutaneous eruption, and by the length of the remission.

It may be distinguished from rheumatism, as it prevails epidemically, and a period of febrile excitement precedes the arthritic phenomena. It may be distinguished from relapsing fever by the eruption and by the character of the remissions.

PROGNOSIS.—The prognosis is always favorable, although the symptoms which attend its development may be alarmingly severe. The prognosis is only unfavorable when it occurs in the very aged or in feeble infants.

TREATMENT.—This fever always runs a definite course, and its treatment is the symptomatic treatment of fever, combined with well recognized anti-rheumatic remedies.

It is claimed that emetics and free purgation diminish the intensity of the fever. A favorite combination is ipecac, calomel, and colchicum—these to be administered every night in cathartic doses. Calomel should never be administered alone, nor in combination with other drugs, if its specific effect is likely to be produced.

The administration of colchicum with spirits of nitre and nitrate of potash, in such proportion that profuse diaphoresis may be produced, in connection with the administra-

tion of effervescing draughts, will usually afford relief from the pain in the head and limbs. Should the arthritic pains still be felt, opium may be administered in sufficient quantity to afford relief.

During the remission the bowels should be kept freely open with saline purgatives, and quinine combined with an alkali should be given at stated intervals. Narcotics may be given in small doses to produce sleep, should the patient be wakeful. By the employment of these measures a return of fever may be prevented and the arthritic pains will gradually subside. If this plan is pursued, should the fever return, it will be mild in character, attended by little constitutional disturbance. The weakness and exhaustion which attend convalescence may be combated by the free use of wine or malt liquors.

The diet should be most nutritious. Nourishment should be administered at stated intervals, during the night as well as during the day.

The lymphatic enlargement, especially in the inguinal region, should be treated locally with iodine.

Citrate of iron and quinine will be found of great service during the convalescing period. If a single joint remains swollen and tender for a considerable period after the subsidence of the fever, the occasional application of a blister is recommended. In some epidemics, relapses after an interval of two or three weeks have been of frequent occurrence. They run a milder course than the primary fever. The relapses more closely resemble an attack of articular rheumatism than they do the primary fever. Quinine is said to furnish great protection against a relapse.

CHRONIC MALARIAL INFECTION.

There is still another form of malarial manifestation closely connected with the subject which has been engaging our attention, of which I will briefly speak. It has been termed malarial cachexia, or better, *chronic malarial infection*. I do not include it in the list of malarial fevers, although it may be a sequela of any form of acute malarial

disease. It may be developed in those who have never suffered from any form of malarial fever, but who have resided for some time in a malarial district. For instance, a person who has had repeated attacks of intermittent or remittent fever, and has become exceedingly anæmic, with an enlarged spleen and enlarged liver, may be regarded as in a condition of chronic malarial cachexia, and is in a condition to present the phenomena of chronic malarial infection. Again, a person who has never had a distinct paroxysm of malarial fever, but who has lived for some time under malarial influences, the malarial poisoning never having been intense, becomes anæmic with enlarged spleen and liver, and presents the phenomena of chronic malarial infection.

MORBID ANATOMY.—The morbid anatomy of chronic malarial infection does not differ from that of the severer types of malarial fever, except in the more advanced stages of the tissue-changes. For instance, the spleen is often ten or twelve times its normal size, tough, firm, and resistent. Its surface is uneven, its capsule enormously thickened, and more or less adherent to the adjacent organs. Its substance is rich in pigment matter, and presents the minute changes either of simple hyperplasia or amyloid degeneration. Similar tissue-changes take place in the liver and kidneys. In some instances the muscular tissue of the heart undergoes fatty or amyloid degenerative changes. Œdema of the subcutaneous cellular tissue, and an accumulation of fluid in the serous cavities, are common attendants of chronic malarial cachexia.

ETIOLOGY.—It is unnecessary to repeat what I have already said in regard to the causes of malarial infection. It may be the result of prolonged exposure in a district only slightly malarial, or of a short exposure in a district strongly malarial.

SYMPTOMS.—Those who are the subjects of chronic malarial infection complain of vertigo, ringing in the ears, loss of memory, disturbance of the sight, loss of appetite, nausea, dyspeptic symptoms, and pain and oppression in the epigastrium. The bowels are rarely constipated ; often

in the morning diarrhoea is present. The sleep is disturbed; it may be profound, but it is unrefreshing. The patient awakes in the morning with a confused feeling about the head and a general feeling of discomfort. Some complain of pains in the back and loins and along the sciatic nerve; others complain of pain and tenderness in the joints and stiffness of the muscles of the limbs and back; they become easily fatigued on exertion, complain of shortness of breath, and have palpitation of the heart.

The nervous system seems to suffer most severely. One of the most common nervous manifestations is local anaesthesia, which usually shows itself upon the outer surface of the thighs. Not unfrequently numbness of the arms and fingers, and tickling and burning of the feet are complained of, and a patient will consult you, thinking he is about to have an attack of paralysis. Last year a prominent lawyer of this city, suffering from chronic malarial infection, came under my observation. Sometimes he would continue an argument in court half an hour after there was a partial loss of consciousness; he would afterwards ask his professional brethren what he had said while in this state.

Hemiplegia sometimes occurs. I remember one case in which there was complete loss of power over the right arm and leg, yet no facial paralysis. This patient had never had a paroxysm of malarial fever, and for that reason the possibility of malarial infection had been excluded. Similar manifestations of chronic malarial infection quite frequently occur in those who have never had a distinct malarial paroxysm.

You may have a form of chronic malarial infection unattended by any nervous manifestations. This form shows itself in catarrhal inflammations affecting the mucous membrane of the stomach, intestines, and bronchial tubes. Patients have a form of bronchitis which is really a chronic malarial affection.

A gastro-enteritis, in which there is marked interference with digestion, may be developed as the result of chronic malarial infection. If this is treated with the ordinary remedies for dyspepsia, no good result is accomplished,

while a few doses of quinine will establish the diagnosis and relieve the patient.

The chronic catarrh of the intestines resulting from chronic malarial poisoning may give rise to a troublesome diarrhœa, which will assume all the characteristics of chronic diarrhœa. As I have already stated, anæmia is a very common result of long-continued malarial poisoning, and palpitation of the heart is a very frequent and sometimes distressing accompaniment of such anæmia. With many persons it gives rise to temporary attacks of melancholia and hypochondriasis. Such persons imagine they have disease of the heart, or kidney, or spine, etc. In some cases the hypochondriasis assumes a suicidal character, or, at least, the individual threatens self-destruction, though I never knew one to do any harm to himself during the attack.

Another nervous manifestation of chronic malarial infection is neuralgia. Certain nerve-trunks or their roots seem to be directly affected, while the nerve-centre connected with the affected nerve-trunks escapes. The first branch of the fifth nerve is most liable to be affected in malarial neuralgia. This neuralgia follows a periodic course. Persons over forty are most liable to be affected by it. Usually the nerve-trunks first affected are the ones involved in successive attacks; for instance, if a certain intercostal nerve is the seat of the primary neuralgic paroxysm, at each subsequent attack this particular nerve will be the seat of the neuralgia.

In some instances chronic malarial infection manifests itself by hemorrhages from the mucous surfaces, such as epistaxis, hæmatemesis, hæmaturia, etc. The most troublesome case of menorrhagia (occurring independent of a mechanical cause) which has come under my observation recovered after the administration of large doses of quinine, when all the remedies ordinarily employed in such cases had failed to produce the desired result.

Recently a patient came under my observation who was in a scorbutic condition, with spongy gums, and with large purpuric spots scattered over the surface of the body; his

surroundings and the effect produced by anti-malarial treatment left little doubt in my mind but that chronic malarial infection was the cause of all the scorbutic and purpuric manifestations.

DIFFERENTIAL DIAGNOSIS.—The first question that now arises is, how can you decide whether these manifestations to which I have referred are malarial or non-malarial? In the majority of cases there will be some enlargement of the spleen—it may be only very slight. There is not necessarily any rise in temperature. The manifestations will be more or less paroxysmal. If the patient has localized anæsthesia or hyperæsthesia, it will be found to be more severe some time in the morning or evening. If he has lost power over one portion of the body, he will find that the loss of power is more marked at a certain period of the day. The patient may not observe this, unless you direct his attention to the fact; then he will readily recognize it. It is for you to elicit the fact by a careful examination.

You will also find in the severer cases of chronic malarial infection, when there is hemiplegia or some structural change affecting the mucous membrane of the stomach, intestines, bronchial tubes, etc., that there are also evidences of pigmentation of the tissues. Free pigment is frequently found in the blood. It is not found in those cases where the malarial poisoning is slight, where it is only sufficient to produce ringing in the ears and slight attacks of neuralgia, perhaps accompanied by slight gastro-intestinal catarrh; but when the malarial poisoning is sufficiently intense to cause temporary loss of consciousness, hemiplegia, or any other of the severer manifestations already alluded to, even though there has been no distinct malarial paroxysm, an examination of the blood will almost certainly give evidence of free pigmentation.

The diagnosis of chronic malarial infection, to a certain extent, depends upon the circumstances which attend its development. If the individual has repeatedly suffered from malarial fever paroxysms, or if he has resided for some time in a malarial district, even though he may not have had a distinct malarial paroxysm, though none of the

phenomena to which I have just referred have been developed, and though that peculiar malarial cachexia which is so characteristic of malarial poisoning is not present, yet it is always well to carefully consider the question of malarial infection.

While the manifestations of chronic malarial poisoning may be called legion—and in many instances they very closely simulate the phenomena of other diseases—still, with a history of possible malarial exposure, by a system of exclusion you reach the fact that the patient is suffering from some form of blood poisoning. When you have reached that conclusion you are able readily to determine the nature of such poisoning. In very doubtful cases you may arrive at a diagnosis, or perhaps confirm an uncertain diagnosis by treatment, in the same way in which we sometimes detect syphilitic infection by the effects of treatment.

PROGNOSIS.—The prognosis in chronic malarial infection depends upon the severity of its manifestations. The degree of enlargement of the spleen and liver is a reliable indication of its severity.

When the symptoms are mild and the spleen is but slightly enlarged, and when neither ascites nor œdema of the lower extremities is present, the prognosis is generally good. If the patient is very anæmic, the spleen very greatly enlarged, and the area of hepatic dulness very much increased, the prognosis is unfavorable. When distinct tumors can be detected in the spleen and liver, they indicate an exceedingly grave form of malarial infection; if the tumors are large, they can rarely be reduced. If the individual in whom these tumors are found removes from a malarial district, a long time may elapse before they apparently very much interfere with his health and comfort.

You must take into consideration the possibility of your patient being able to take up his permanent residence in a non-malarious region, before you make a prognosis in any given case.

So long as such a patient is under malarial influences, however slightly malarial they may be, the progress of the disease cannot be permanently arrested; and when the

manifestations of the graver forms of malarial infection are present, there is little prospect that the disease can be temporarily relieved so long as the patient remains in the malarial district.

TREATMENT.—The first and most important thing to be accomplished in the treatment of chronic malarial infection *is the removal of the individual from a malarious district to a high, warm, mountainous region.* It is of the greatest importance that all exposure to wet and cold, and the damp air of the evenings and nights, should be avoided; the sleeping apartments must be dry and airy, and flannel should be worn next to the skin.

So long as the thermometer shows even a slight febrile movement, quinine must be given in full doses. If anemia is present, which is usually the case, iron must be combined with the quinine, and administered immediately before or after taking food.

In those cases in which the spleen and liver are very much enlarged, when no febrile excitement is present, *iodide of iron combined with cod-liver oil will be found of great service.*

It is claimed by some that muriate of ammonia has a very beneficial effect in this class of cases, but my own experience does not lead me to favor its use.

If the bowels are constipated, aloes or rhubarb should be given in connection with some of the chlorine mineral waters. In those cases in which the measures already referred to fail to produce any improvement or afford any permanent relief, arsenic may be resorted to, but the effects of the drug must be carefully watched, and on the appearance of œdema or of gastric disturbance, it must be promptly discontinued. It must be borne in mind that the use of all these therapeutic agents is not sufficient; proper attention must be paid to hygienic measures.

The neuralgias which are such frequent manifestations of this infection are best treated by combining a full dose of opium with large doses of quinine. If paralysis is a manifestation of the malarial poisoning, strychnine, iron, and quinine may be combined in its treatment, in con-

nection with cold douches and friction to the paralyzed limbs.

A most nutritious diet and a liberal use of good wine is indicated in all cases of chronic malarial infection. The daily use of brandy in small quantities is of great service.

I will add a few words in regard to the use of quinine in this class of cases. I am convinced that the indiscriminate use of this drug often does harm. After fairly testing its effects, if no relief is obtained, its use should be discontinued for a time, or at least until the beneficial effect of a removal from a malarial district is tried, or until, by the use of mild cathartics and the daily administration of cod-liver oil and iron, the patient is in a condition to be benefited by it. Quinine seems to have no effect upon many persons suffering from the severe manifestations of this infection, so long as they remain in a malarial district. It is of the greatest importance that you should early make yourself familiar with the condition in which quinine is indicated in the treatment of this class of affections. Let me impress upon you the importance of avoiding depressing remedies in all forms of chronic malarial infection. Drastic cathartics, exhausting diaphoretics, and all other depressing remedies must be carefully avoided. They do great harm by exhausting the already enfeebled vital powers. Especially is this true in regard to the free use of mercurials, which are so commonly resorted to in their management. Unquestionably, an occasional cathartic dose of calomel is of service, but the administration of small doses repeated after short intervals, in order to produce the constitutional effects of the drug, will always be followed by the more serious manifestations of the malarial infection.

The exhausted system of this class of patients needs rest, concentrated nutrition, and the supporting influence of a change of climate and tonics.

LECTURE XVI.

TYPHO-MALARIAL FEVER.

Introduction.—Morbid Anatomy.—Etiology.—Symptoms.

I SHALL this morning commence the history of typho-malarial fever. I have included this fever in the list of the malarial fevers, although it is not altogether malarial in its origin; malarial poison, however, is so essential to its development that it may very properly be regarded as one of the malarial fevers.

As its name indicates, it has many elements in common with typhoid, and many which ally it to remittent fever. To the term "typho-malarial" different significations have been given by different observers. By one class the term has been employed to indicate the presence of malaria, and also the specific poison which produces typhoid fever.

By another class of observers the term has been employed to indicate the presence of malaria, and also a *septic* poison which differs from the specific poison that gives rise to typhoid fever.

There is still another class of observers who doubt the existence of such a form of fever, and regard the so-called typhoid element as nothing more than a "typhoid condition," liable to be developed in connection with remittent fever, as well as many other diseases.

The term typho-malarial is a convenient one for the first class of observers, and is one which can be employed by them without confusion; whereas, for the second class of observers, it is exceedingly inconvenient, giving rise to con-

fusion, because it does not embrace the views held by them regarding the etiology of the disease.

But we have the term, and I shall employ it as one denoting a fever which is produced by the combined action of a *septic* and a *malarial* poison. As far as possible I shall use the word *septic* when speaking of the poisons which are associated in the production of the disease, and the term *typhoid* will be reserved for that peculiar condition known as the "typhoid condition," and for the specific disease known as typhoid fever. You will meet with some cases of typho-malarial fever in which the septic element predominates, and others in which the malarial element is predominant. The preponderance of the leading features of the one or the other of these two forms of fever will enable you to determine with a good degree of certainty the course, prognosis, and treatment of each individual case. The distinguishing lines, however, between these two elements are not always sharply defined, but almost imperceptibly the symptoms dependent upon one poison become mingled with those developed by the other. Both of these elements may be modified in their manner of development and in their morbid anatomy, by the occurrence of various intercurrent complications, such as scurvy, pneumonia, etc.

MORBID ANATOMY.—The changes which take place in the constituents of the blood in typho-malarial fever, so far as we are yet able to determine, are similar to those which occur in typhoid fever, combined with those which are characteristic of malarial fever; the presence of free pigment granules in the blood is often a strong point in its differential diagnosis.

In connection with these blood changes, there are more or less parenchymatous changes in the internal organs similar to those met with in other forms of fever and in acute infectious diseases. The *liver* is increased in size, and its cut surface presents an appearance which closely resembles that known as nutmeg liver. Sometimes it presents the peculiar bronzed color of the liver in remittent fever; at other times it very closely resembles the liver of yellow fever. A microscopical examination shows free fat and more or less

brown pigment granules in the hepatic cells. In most cases of this fever the *spleen* is enlarged, softened, and of an almost black color. The Malpighian bodies are prominent, and present the appearance on the torn surface of the spleen of little tumors, which vary in size from a pin's head to that of a pea. The organ is rarely as much enlarged or softened as in typhoid or remittent fevers. It is always the seat of more or less pigmentation.

No uniform change will be noticed in the *kidneys*, except that of hyperæmia, which will be most marked in their cortical substance.

The *lungs* at their most depending portion are the seat of more or less extensive hypostatic congestion. Splenization of the lungs is not as frequently present as in typhoid fever.

The *heart* is pale and flabby. Its muscular fibres are the seat of a granular degeneration similar to that which takes place in the heart in typhoid fever. Exsanguinated clots more or less firm may be found in its cavities, but they have nothing peculiar about them. They closely resemble those found in persons who have died from failure of heart power. They are rarely, if ever, the direct cause of death. My own examinations of the intestinal lesions of this fever have led me to adopt, for the most part, the descriptions which have been published by Dr. J. J. Woodward, of the U. S. A. In fact, Dr. Woodward's investigations in this direction may be regarded as exhaustive. That the intestinal changes of typho-malarial fever very closely resemble those of typhoid fever there can be no question; by some they have been regarded as identical, but I think, if we very carefully observe them, some very marked differences can be recognized; especially if we attempt to divide the stages of their development into periods so as to correspond to the days and weeks of the fever, as is possible with the intestinal changes of typhoid fever.

As in typhoid fever, the principal and almost constant changes are to be found in and around the closed follicles of the intestinal tract. These changes are made manifest by the gradual enlargement of the follicles, which, as they enlarge, become more or less pigmented.

At the post-mortem examination of one who has died of this fever, you will usually find these glands in all stages of this pathological process, from slight enlargement and softening to ulceration of the entire follicle. The summit of the enlarged follicle is the first seat of the ulcer. These ulcers may involve a single follicle, or they may invade the adjacent mucous membrane, and produce ulcers from one-half an inch to an inch in diameter. The largest and most extensive ulcerations are to be found in the ileum and involving the Peyerian patches. The edges of these ulcers are irregular and everted; their base is usually of a grayish color, often mottled with black points. These ulcers may extend into the submucous tissue and involve the muscular coat of the intestine, and even perforate the peritoneal covering of the intestines.

In the earlier stages there is little to distinguish these intestinal changes from similar ones which develop in typhoid fever, except, perhaps, the tendency to the deposit of black pigment in the enlarged follicles. In a later stage, certain peculiarities are present, which are often sufficiently distinctive to designate the case as one of typho-malarial fever. For instance, in typho-malarial fever there is a gradual elevation of the mucous membrane surrounding the enlarged follicles, which, if ulcers exist on their edges, reaches a thickness of from three to six lines.

These ulcers differ from those of typhoid fever in that the enlarged patch rises abruptly from the mucous membrane, and in such a manner that the summit is often larger than the constricting base. Besides, the umbilical depression so often seen in ordinary typhoid patches prior to ulceration is rarely observed in typho-malarial fever. As I have already stated, the ulcers in typho-malarial fever present ragged, irregular edges, which are usually extensively undermined, in consequence of the erosions extending into the submucous tissue, rather than into the glandular layer of the mucous membrane. This undermining of the edges is much more extensive than in typhoid ulcers.

The mucous membrane between the follicles presents the

ordinary appearance of catarrhal inflammation, namely, there is more or less congestion, tumefaction, and in the later stages thickening and softening of its tissue.

The minute anatomical changes which attend the development of these intestinal lesions, as determined by the microscope, do not essentially differ from those which I have already described as occurring in typhoid fever, except that they have no regular stage of development marked by days and weeks, the processes are slower in their development, and the presence of pigment in the enlarged and ulcerating follicles stamp it as depending upon an essentially different exciting cause. Hence, although the intestinal lesions of this fever very closely resemble those of typhoid, they are not identical, but evidently belong to another type of disease. Undoubtedly, there are cases in each of these two forms of fever between which, by the intestinal lesions alone, it is impossible to draw the line of distinction: but in typical cases this is easily done.

Intestinal perforation, and a consequent peritonitis, the result of the intestinal ulceration, may occur in typhomalarial fever, but you will rarely meet with such an accident. Usually the mesenteric glands are more or less enlarged, and in the advanced stages of the disease more or less softened. They are of a livid color, and more or less pigmented. The greatest enlargement of these glands will be found in that portion of the mesentery which corresponds to the most extensive and advanced intestinal changes.

The principal changes in the structure of the glands are similar to those which occur in a purely inflammatory process.

Occasionally, minute ulcers are met with in the mucous membrane of the stomach and large intestines, and the mucous membrane of the stomach is not unfrequently very greatly softened, and the mucous membrane of the large intestine, if there have been any manifestations of scurvy during the progress of the fever, will be thickened and softened, perhaps extensively ulcerated, presenting an appearance, in some instances, closely resembling those found after death in chronic malarial dysentery. While, there-

fore, we find no pathological lesions which can be regarded as characteristic of this type of fever, and while the lesions which we do find very closely resemble those of typhoid fever on the one hand, and remittent fever on the other, still there are marked differences which distinguish it from either of these fevers sufficiently to stamp it as a distinct type of fever.

ETIOLOGY.—It is difficult to determine the true etiology of typho-malarial fever. That malarial poison is necessary for its development there can be no question. It is equally certain that some other poison besides malaria is in operation whenever this fever prevails. That this poison is not the specific poison of typhoid fever is apparent from the fact that its development and spread, as far as can be determined, is in no way connected with the excrements of one suffering from this fever.

There are a few facts connected with its development which are now well established :

First.—It is only met with in malarial districts.

Second.—In the majority of instances, when this fever has prevailed, its development has been preceded or attended by marked, and easily recognized, anti-hygienic conditions, such as overcrowding, bad sewerage, and other conditions favorable to the development of septic poison.

Third.—That it is a *non-contagious disease*, and is never propagated from the affected to the healthy, either directly by personal contagion, or indirectly by morbid excretions.

Fourth.—In its morbid anatomy and symptomatology it is a combination of two well recognized forms of fever. The special symptoms and lesions of one or the other of these fevers stamp its character, and indicate its alliance to a malarial or septic type of fever.

In large cities, in which malarial diseases are prevalent, *sewer gases seem to furnish the septic element which is so necessary for the development of this type of fever*. The history of disease in our own city during the past few years furnishes striking examples of the combination of these two poisons in developing a type of fever which it seems to me must be classed under this head.

SYMPTOMS.—It is even more difficult to present a typical picture of this fever than of typhoid. To give you even an outline of its symptoms which shall be approximately true of all, or even the majority of cases, is impossible. Its clinical history varies as the malarial or septic element predominates. Besides, there are a large number of cases in which neither of these elements can be said to predominate, for the patient almost insensibly passes from a malarial into a typhoid condition. There are also certain anti-hygienic conditions which may be present, which give to the fever an unusual and peculiar type. For example, when those conditions exist which favor the development of scurvy, if typho-malarial fever is prevailing, as the patient enters upon the second week of the fever the scorbutic phenomena will become prominent.

At times the dysenteric element may be engrafted on this fever, which shall greatly modify its course, and lead to a train of symptoms and morbid changes which shall very closely ally it to epidemic dysentery.

The course of this fever may also be greatly modified by certain local complications which are especially liable to occur during the second or third week. The presence of any of these conditions will greatly change its clinical history, but the phenomena which attend its early development will always be sufficient to determine its true character.

In considering in detail the symptoms of this fever, I will first describe that class of cases in which the *malarial element is predominant*.

This type of fever is usually ushered in by a distinct chill. In some instances no premonitory symptoms are present, in other cases the chill is preceded by wandering pains in the limbs and back, headache, loss of appetite, and a feeling of great exhaustion. In a large proportion of cases, in the early stage, the countenance has a peculiar waxy, clay-colored, or yellowish tinge. The chill varies in duration from half an hour to an hour, and in character closely resembles the chill of simple remittent fever. It is immediately followed by active febrile symptoms, the temperature rising in a few hours to 103° F. or 104° F. The pulse

reaches 100, and is full and forcible. The excretions are all checked, and there is mental disturbance and sometimes delirium. When once established, the fever pursues a variable course. At its onset, and for the first few days, its phenomena often closely resemble those of simple remittent fever, though the remissions are never so well defined as in remittent, and there is at the very onset of the fever an amount of intestinal disturbance which is rarely present in simple remittent. The existence of abdominal tenderness, especially in the right iliac fossa, is a strong point in the differential diagnosis of typho-malarial and simple remittent fever in favor of the former. As the temperature rises, nausea, vomiting, and epigastric tenderness are present in a greater or less degree. These gastric symptoms bear a close resemblance to those which attend the development of remittent fever, while the intestinal and abdominal symptoms are similar to those of typhoid fever. Diarrhœa may precede the chill; in most cases it is present during the period of fever. At first the tongue presents a pale, flabby appearance, with a smooth surface; soon it becomes covered with a white or yellowish-white coating; later it becomes red and the coating becomes brownish; in severe cases it may suddenly become clean, red and shining, and sordes may collect upon the teeth and lips.

In those cases in which a scorbutic element exists, the tongue is enlarged, pale, and flabby, its surface smooth and covered with a white fur, which is thickest on its edges, the gums are swollen and present the characteristic appearance of scurvy.

In those cases in which a dysenteric element is present as the fever develops the dysenteric symptoms become prominent, the discharges from the bowels are blood-stained and watery. The tongue soon becomes dry and brown, and the patient shows signs of extreme exhaustion, with few of the gastric symptoms which are usually so well marked in the early period of the fever.

Throughout the whole course of the disease there is a marked tendency to periodicity, the exacerbations usually assuming a tertian type. In fatal cases, as the patient

reaches the second or third week, the symptoms are very like those of fatal typhoid fever: the prostration becomes more and more complete, the pulse reaches 130 or 140, is feeble and irregular, the patient gradually passes into a state of stupor and coma, involuntary evacuations take place, and death ensues.

In cases that recover, symptoms of amendment may be noticed between the tenth and twentieth days. The tongue begins to become clean, the abdominal symptoms subside, the pulse becomes less frequent and fuller, the disturbance of the nervous system disappears, the appetite gradually returns, and the patient enters upon a tedious convalescence, which is attended by more or less diarrhœa, mental stupor, cardiac irritability, and a slow return of mental and physical vigor.

The train of symptoms thus briefly sketched may be greatly modified by a variety of complications. Not unfrequently pulmonary complications develop during its second week, and so change its phenomena that the fever element may be overlooked and the pulmonary element alone engage the attention of the physician.

Suppurative inflammation of the cervical and inguinal glands sometimes complicates this type of fever, and leads one to the mistake of regarding it as purely a suppurative fever.

Again, scurvy under certain anti-hygienic conditions may so modify the usual phenomena of typho-malarial fever, that it has led some to regard this fever when developed under such circumstances as an entirely new type of fever, entirely losing sight of its malarial element, and classing it among the infectious fevers. The scorbutic element in this class of cases is developed in connection with the malarial exposure.

LECTURE XVII.

TYPHO-MALARIAL FEVER.

Symptoms (continued).—Differential Diagnosis.—Prognosis.—Treatment.

I HAVE mentioned the prominent symptoms which attend the development of that type of typho-malarial fever in which the malarial element predominates, and will now speak of those present in the *septic type* of this fever. Although the premonitory symptoms of this type, such as lassitude, headache, pains in the back and limbs, resemble those of typical typhoid fever, either a distinct chill or a complete intermittent or remittent paroxysm ushers in the febrile symptoms.

The rise in temperature following the ushering-in chill has no typical range; in some cases the rise is gradual, not reaching its maximum before the middle of the second week; in other cases the rise is sudden, reaching 104° F. or 105° F. within twenty-four hours after the occurrence of the chill. Throughout the whole course of the fever the same tendency to periodicity exists which was noticed in the malarial type of this fever.

In typhoid fever, during the first week, there are indistinct forenoon remissions and afternoon exacerbations, but in this fever the remissions are well marked, especially on every second or third day, causing the fever to assume a more or less distinct tertian or quartan type. One of the earliest symptoms is well-marked hepatic tenderness; with

the hepatic tenderness there is enlargement of the spleen, which, as the fever progresses, reaches a much larger size than is ordinarily met with in typhoid fever. During the first week the pulse is full and rarely more than 100 beats per minute, but during the second and third weeks it is small and compressible, and in severe cases intermittent, and ranges from 110 to 130 per minute. The appearance of the tongue varies with the period of the fever. At first it is swollen, with red projecting papillae, and has a light white coating. As the typhoid condition becomes more prominent its appearance changes; it becomes dry and brown, and frequently the brown coating cracks, and fissures are formed in the mucous membrane underneath. Should the tongue become moist and begin to clean, you may regard convalescence as established. The coating is removed in two ways, either gradually from the edges to the centre, or it is thrown off in flakes. In the latter case, after the removal of the coating, the tongue assumes a beefy red appearance, and after a short time may again become brown and dry. Under such circumstances there will be a renewal of the fever-symptoms.

After the fever has continued a few days the surface becomes dry and harsh, and the skin assumes a bronzed hue, which is quite characteristic of this fever; sometimes, instead of this bronzed hue of the surface, there is well-marked jaundice.

The changes in the urine do not differ from those which usually attend febrile excitement. The urine gradually diminishes in quantity and deepens in color until convalescence commences, when it increases in quantity until convalescence is reached. It is rarely albuminous.

Diarrhoea may occur at any period. It is not usually excessive until the second or third week. There is nothing characteristic about the discharges. They are usually of an exceedingly fetid odor, watery, and dark-colored; in the later stages of the disease they sometimes contain blood. In some instances the character of the stools is termed bilious, and an excessive hepatic secretion is then indicated; at other times they are of a dark clay color, showing a de-

fiency of the biliary secretion. With the diarrhœa there is usually more or less abdominal tenderness, especially in the right iliac region; but the tympanitis, which is so constant an attendant of typhoid fever, is rarely well marked in typho-malarial fever. In many cases there is retraction of the abdomen.

As I have already stated, headache is a very constant and prominent symptom in the early period of this fever. It often precedes the ushering-in chill. As the fever progresses it gives place to a delirium, which is never violent, but which is muttering in character, and is attended by restlessness and insomnia, or by drowsiness, subsultus, picking at the bed-clothes, and great nervous prostration. If delirium is not present, or after it has disappeared during convalescence, there is great lack of mental vigor and a tendency to mental sluggishness. The other nervous phenomena, which are usually present in any condition when marked typhoid symptoms exist, are not prominent in this fever. The subsequent phenomena which may attend its development will vary with the intensity of the fever and the resisting power of the patient.

In fatal cases, towards the close of the second week, symptoms of extreme prostration come on, the patient gradually passes into a state of stupor, which lapses into one of coma, and death ensues.

In cases that are to recover, by the end of the second week the tongue begins to clean, the gastric and intestinal symptoms, with the exception of the diarrhœa, begin to subside, the pulse becomes slower, the nervous disturbances disappear, the appetite returns, and the patient enters on a convalescence which is usually protracted.

It is apparent that the early stage of this fever very closely resembles that of simple remittent, while its latter stage as closely resembles that of typhoid.

The phenomena of both stages may be modified by certain anti-hygienic surroundings, to which those suffering with this fever may have been subjected prior to, and during, its development. Thus, when it prevails among those who have suffered privations, been badly fed, badly

clothed, overcrowded in badly ventilated apartments, surrounded by decomposing animal and vegetable substances, although the fever is attended by the same general phenomena which characterize the typhoid type, there are certain variations which ally it to relapsing fever. Prominent among these are neuralgia and arthritic pains in various parts of the body, especially in the back and limbs; hemorrhagic tendencies, marked by bleedings from the gums, mucous surfaces; and not unfrequently large ecchymoses occur in various parts of the body. In this class of cases from the commencement the fever is of low type, with quotidian exacerbations and remissions. Diarrhœa usually precedes the development of the febrile symptoms. Frequently during the second week a muttering delirium comes on, accompanied by drowsiness and a tendency to stupor. Dependancy, indisposition to make any exertion, and a state of utter indifference as to the future, is frequently met with during the entire period of the fever.

In fatal cases death may be the result of hemorrhage from the mucous surfaces, or from exhaustion. In this class of cases there is great irritability of the heart and a peculiar mental and physical prostration.

In cases that recover, convalescence comes on late, and is slow and tedious. Diarrhœa frequently follows the subsidence of the fever, which in many cases cannot be controlled, and leads to a fatal result.

The complications which may modify the course of any variety of typho-malarial fever are very similar to those which are met with in typical typhoid fever. Of these the most frequent is inflammation of the respiratory organs, the development of which is marked by those symptoms which usually attend the development of the different acute pulmonary affections. In the majority of instances the signs of bronchitis are not present until the fever is well established. The bronchitis resists treatment, and does not disappear until convalescence is fully established. When pneumonia occurs it is catarrhal in character, and few of the strongly marked rational symptoms of ordinary pneumonia are present. The physical signs, however, will

always enable you to determine the presence of pulmonary complications, and any great irregularity in temperature during the course of the fever should lead you to make a careful physical examination of the chest.

It is sometimes difficult to distinguish between the cerebral symptoms of this fever and those symptoms which attend meningeal complications, but the meningeal complications are of so very rare occurrence that it is safe to assume they are not present until some of the diagnostic symptoms of meningitis occur.

We rarely have serious abdominal complications, such as intestinal perforation, peritonitis, and hemorrhage, but when they do occur their advent is marked by such urgent symptoms that one loses sight of the ordinary symptoms of the fever.

It is hardly necessary for me to refer to those modifications in the clinical history of this fever which follow the development of abscesses, bed-sores, gangrene, etc.

DIFFERENTIAL DIAGNOSIS.—The affections with which typho-malarial fever are likely to be confounded are typhoid, remittent, relapsing, typhus, and yellow fever.

The septic type of typho-malarial fever, in many of its phenomena, so closely resembles typhoid fever that frequently it is difficult to make a differential diagnosis. I will briefly state the points of difference in their clinical history.

The advent of typho-malarial fever is usually marked by a distinct chill, while typhoid comes on insidiously, and is not attended by a distinct chill, but by a chilly sensation. The rise of temperature in typho-malarial fever is sudden and follows no typical range, while in typhoid the typical range of temperature during the first week is almost diagnostic of the fever.

In typhoid fever, on the sixth or eighth day, rose-colored spots appear; these are a distinctive mark between it and typho-malarial fever. Although in the latter an eruption may be present, yet it has none of the characteristics of the typhoid eruption, is not rose-colored, does not disappear on pressure, and remains visible throughout the whole course of the fever.

Besides the absence of these characteristic symptoms of typhoid fever, in typho-malarial fever we have a distinct periodicity in the febrile action, a certain icteroid hue of the skin, hepatic tenderness, extensive splenic enlargement, and great gastric disturbance; conjoined with these the appearance of the tongue, the character of the diarrhoea, and the non-infectious character of the stools in typho-malarial fever serve as important aids in the differential diagnosis of these two forms of fever. In typho-malarial fever, upon microscopical examination of the blood, we find free pigment; this is never or rarely found in the blood in typhoid fever.

The malarial type of typho-malarial fever resembles remittent fever in its ushering-in symptoms. In both cases there is a chill followed by fever, attended by one or more distinct exacerbations and remissions. The early appearance of the enteric symptoms, attended by other well-marked typhoid phenomena by the end of the second week, establishes the diagnosis of this type of malarial fever, and as the fever progresses the typhoid condition becomes more and more apparent. Besides, remittent fever yields more promptly to the use of quinine than does typho-malarial fever.

Severe cases of typho-malarial fever, which are complicated by scorbutic tendencies, marked by petechiae and great prostration of the vital powers, may be confounded with typhus fever; yet the severity of the attack, the higher range of temperature, the greater frequency of the pulse, the dusky countenance, the absence of diarrhoea and all other abdominal symptoms in typhus fever, renders it easy to make the differential diagnosis between the two types of fever. Besides, typhus fever has a characteristic eruption, is only propagated by contagion, and if it prevails, does so epidemically. Occasionally yellow fever has been confounded with typho-malarial fever, and on this account I will mention some of the prominent diagnostic symptoms of yellow fever which distinguish it from typho-malarial fever.

The range of temperature is lower in *yellow* than in

typho-malarial fever, and on the third or fourth day it falls suddenly, and there is more or less complete remission. The circumorbital pain, the appearance of the eye, the peculiar color of the skin, the character of the matter vomited, the absence of diarrhœa, the presence of albumen in the urine, and the shorter duration of the disease, will enable you to make the diagnosis of yellow fever. Again, yellow fever usually prevails epidemically, and is confined to certain localities and certain seasons of the year. It is a portable disease, and the yellow fever poison may be conveyed from an infected to a non-infected district by means of clothing or merchandise, while the poison of the typho-malarial fever is of endemic origin, and cannot be carried beyond the infected district.

The points of differential diagnosis between typho-malarial and relapsing fever will be considered under the head of relapsing fever.

The differential diagnosis between cerebro-spinal meningitis and typho-malarial fever is sometimes attended with great difficulty.

PROGNOSIS.—The ratio of mortality in typho-malarial fever varies greatly in the different regions in which it occurs, and as the malarial or septic element predominates. The hygienic surroundings of the patient and the range of atmospheric temperature will also very greatly influence your prognosis. Statistics of this fever in different localities and in different years give the ratio of mortality from one in twelve to one in twenty-four. The septic type is more fatal than the malarial type. Great caution should be exercised in prognosticating the result of any case, for the apparently mildest cases sometimes suddenly assume a severe type and terminate fatally, while very severe and apparently hopeless cases unexpectedly improve, and recovery takes place.

The average duration of those cases which terminate in recovery is from three to four weeks; the duration varies with the different types of the fever. In the malarial variety the duration is always shorter than in the septic. The period of convalescence is prolonged; three or four

weeks often elapse before the patient is completely restored to health. A fatal relapse may occur at any period during convalescence. In those cases that terminate fatally, death most frequently occurs during the second or third week ; it may occur as late as the close of the sixth week.

The occurrence of any of the complications to which I have referred as possibly taking place during the course of this fever will very materially influence the prognosis in any given case. Capillary bronchitis and pneumonia are especially dangerous when they develop during the third week of the fever.

Anti-hygienic surroundings, such as overcrowding and improper food, materially affect the prognosis. If typho-malarial fever prevails among those who are crowded into badly-ventilated apartments, who from filth and improper nutrition have septic and scorbutic tendencies, the ratio of mortality is much greater than among those who are free from such complicating influences.

The symptoms which may be regarded as indicating an unfavorable termination are : a continued high temperature, showing little or no tendency to remission ; a very frequent, feeble, fluttering pulse ; profuse diarrhœa, the discharges at times being involuntary and containing mucus, pus, and blood ; a dry, red, cracked and fissured tongue ; great drowsiness, with a tendency to stupor and coma, and the appearance of petechial spots on the surface of the body, attended by frequent hemorrhages from the lips, gums, and tongue. In a severe case, the occurrence of any of these complications renders the prognosis more unfavorable. The character of the prevailing fever will also greatly influence the prognosis in any given case. If the type of the prevailing fever is mild, or if comparatively few deaths have occurred, though the symptoms in a given case may appear unfavorable, yet recovery is probable. If, on the other hand, the type is severe, and many deaths have occurred, apparently mild cases will suddenly become severe, and the prognosis becomes unfavorable.

As I have already stated, the hygienic surroundings and the previous habits of the patient very greatly influence the

prognosis. With drunkards, and those enervated by vicious habits, a mild type of this fever will probably prove fatal.

TREATMENT.—The treatment of typho-malarial fever varies with its type. No plan can be presented which will be applicable to all cases.

As in other forms of disease, the first question that meets us under the head of treatment is, cannot the development of this fever be prevented? While speaking of its etiology, I stated that its development was principally due to three causes—namely, malarial poison, overcrowding, and improper diet. In a large proportion of instances it is possible to do away with the last two causes. The overcrowding and the faulty diet may be prevented, and thus the septic poison which gives to this fever its typhoid type may be destroyed or its development prevented. The strict observance of hygienic laws in the localities where this fever prevails has in some instances entirely changed the type of the disease. Even after the fever symptoms have been well developed, the removal of patients from anti-hygienic surroundings has frequently been attended by the most satisfactory results. When isolated cases of this fever are met with in localities apparently free from such sources of infection, a careful search should be instituted, in order to find the source of the infection. Defective sewerage and faulty drainage have been found to be fruitful sources of infection.

The therapeutic measures which may be employed in the treatment of this form of fever vary with the type of fever and the peculiarities of each individual case. There are no specifics.

In those cases in which the malarial element predominates, the administration of quinine as an antiperiodic will produce the desired result, and in many instances arrest the progress or shorten the duration of the fever; but in those cases in which the septic element predominates, while quinine may act as an antipyretic in the same way as it does in typhoid fever, it has little power to arrest the progress or shorten the duration of the fever, but it will, in most instances, render the course of the fever milder.

In those cases in which the malarial element predomi-

nates, which are ushered in by distinct chills, followed by one or two distinct remissions and exacerbations, during the first remission twenty or thirty grains of quinine, in two or three doses of ten grains each, should be administered every hour until the desired quantity has been given. If it is promptly and freely administered, it seldom fails to produce a beneficial effect; usually the febrile exacerbations will not return, or if they do they are less severe, and in a few days entirely disappear.

In those cases which begin more insidiously and are developed more gradually, if there is a distinct periodicity to the febrile phenomena, without distinct remission, although, by the administration of quinine, you may not shorten the duration of the disease, yet the fever will run a modified and very much milder course.

If the first full doses of quinine fail to produce any effect in this class of cases, its administration in moderate doses, perhaps ten grains twice a day, must be continued for several days before it will markedly modify the severity of the fever. In no type of the fever does the quinine exert any specific influence except over the malarial element; the enteric phenomena are either not at all, or only indirectly, modified by the antipyretic power of the drug. Hence, it is apparent that in those cases in which the malarial element is slight, and in which the septic element is prominent, while quinine fails to exercise any controlling influence over the progress of the fever, it will mitigate its severity, and act more powerfully as an antipyretic than it will in any other form of continued fever.

It has been claimed by some that arsenic has a specific influence over typho-malarial fever, and that it exercises a peculiar and most beneficial effect upon the intestinal lesions, materially shortening the duration of the fever. There is little doubt but that arsenic, like quinine, acts beneficially in many cases of the malarial type of this fever; but unquestionably this beneficial effect is due to its acknowledged power over malarial affections, and not to any specific influence which it has over the fever. As an anti-periodic it is inferior to quinine.

The antipyretic treatment of typho-malarial fever does not materially differ from that recommended for the reduction of temperature in typhoid fever. It is of importance to remember that this class of patients do not bear well the prolonged application of cold to the surface, either by means of the cold bath or the cold pack, and that, unless the antipyretic power of quinine is added to the application of cold, very little benefit will be obtained from its employment. The danger resulting from the injudicious use of cold baths is greater in this than in any other infectious disease.

The rules for the administration of stimulants in typho-malarial fever are the same as those given for their administration in typhoid fever. The effects of the first few doses should be carefully watched. They should never be given indiscriminately, for there is greater danger of over-stimulating in this than in any other fever. Their use is indicated whenever signs of heart-failure are present, such as a feeble pulse and an indistinct first sound of the heart. No fixed rule can be laid down as regards the quantity to be administered in any given case; it will vary with the type of the fever and the previous habits of the patient; it should always be administered at stated intervals. The period of the fever at which stimulants should be commenced will also vary. In some cases, stimulants are never required, while in other cases, from the very outset of the fever, they are demanded. In the majority of cases their use is not indicated before the end of the second week. It must be borne in mind that alcohol is not a specific, curative agent in this fever, but that the object of its administration is to sustain the heart and prevent the vital powers from falling below the point at which reparative processes are possible. The use of stimulants is not necessarily contra-indicated when delirium is present. Frequently after their administration the delirium will pass away, and only when it is decidedly increased by their use should they be abandoned.

The state of the bowels, skin, and kidneys demands the closest attention. If, early in the disease, the bowels are

constipated, a calomel purge combined with ten or fifteen grains of quinine will often be followed by marked benefit. In any stage of the disease brisk purgation should be avoided. If diarrhœa is present, it should not be interfered with unless it becomes exhausting; then it should be checked by small doses of opium combined with astringents.

When the skin becomes dry and parched, if cold baths or packs are not admissible, the surface should frequently be sponged with tepid water. It has been proposed by some to apply oil to the surface two or three times every day, when, from extreme exhaustion or any other cause, bathing or sponging of the surface cannot be practised.

Special notice should be taken of the quantity and character of the urine. If it becomes scanty and high-colored, or if there is a temporary suppression, it is of the utmost importance that the functions of the kidneys should be immediately restored. This can be best accomplished by the administration of digitalis combined with spirits of nitre. Sometimes retention may be mistaken for suppression of urine, unless a careful examination be made as to the condition of the bladder. Symptoms referable to disturbance of the nervous system sometimes require special treatment. If there is extreme restlessness, muscular twitchings, or active delirium, opium may be administered in full doses. The effect of the first dose must be carefully watched. If sleep soon follows its administration, and the delirium gradually subsides without any aggravation of the other symptoms, its use may be continued; if, instead of producing sleep, the patient becomes more wakeful, and the delirium is increased and more active, and the other symptoms are greatly aggravated, its use must be immediately abandoned. Under these circumstances chloral may be tried with great care.

Some claim that spirits of turpentine in the treatment of this form of fever has almost a specific power, while others regard it useful only as a stimulant. My own experience leads me to employ it only as a stimulant during the second and third week of the disease, when there is great prostra-

tion and marked typhoid symptoms. It may be given as an emulsion in doses of twenty drops every two hours.

The *diet* best suited to patients with this fever is milk administered in the same way as was proposed in the case of typhoid fever patients.

Special complications occurring during typho-malarial fever must be met with such remedies as the condition of the patient and the peculiar complications may require.

CONTAGIOUS FEVERS.

LECTURE XVIII.

TYPHUS FEVER.

Introduction.—Morbid Anatomy.—Etiology.

AT my last lecture I completed the history of malarial fevers.

I will now commence the history of the *contagious fevers*; and the first which will engage our attention in this class is typhus fever. This fever, like those which we have just been considering, depends upon changes produced in the blood by a morbid agent developed exterior to the body.

Although it has many phenomena in common with the miasmatic contagious fevers, and has until quite recently been classed with typhoid fever, yet with our present knowledge it must be regarded as a distinct type of fever, dependent upon a specific poison, with certain pathological and etiological phenomena which distinguish it from all other forms of disease.

Typhus fever is an epidemic disease. It has received a great variety of names, such as "*ship-fever*," "*hospital fever*," "*jail-fever*," "*camp-fever*," "*pestechial fever*," "*putrid fever*," "*continued fever*," and *typhus fever*. The Germans describe an *abdominal* and *cerebral* typhus. Their abdominal typhus corresponds to our typhoid fever, and their cerebral typhus is our typhus fever.

MORBID ANATOMY.—I shall first consider those pathological lesions which are common to typhus and typhoid fever, and as I draw the line of distinction between them,

you will notice that in many respects the difference is one of degree rather than of kind.

First, I will speak of the changes in the blood.

Blood.—The blood in typhus fever is darker in color than normal, and when drawn from the body during life coagulates imperfectly or not at all; if a clot is formed, it is of the consistency of putty. The fibrin is diminished, or to a greater or less extent loses its coagulating power. At first the red globules are increased in number, but as the disease progresses they diminish in number, the salts of the blood are also changed, and urea and ammonia are present in excess; by some the latter is supposed to be produced by the decomposition of the former. The blood of a typhus fever patient, when drawn from the body, rapidly undergoes ammoniacal decomposition. When the blood is examined microscopically, many of the red blood-globules will be seen to have lost their normal outline, and their edges to have become serrated and irregular. In some instances they will be found to have undergone degeneration; their coloring matter will then pass through the walls of the blood-vessels and stain more or less deeply the tissues and effusions which may have taken place in the serous cavities. These blood-changes are very similar to those which take place in the miasmatic contagious fevers—they differ rather in degree than in kind.

PARENCHYMATOUS DEGENERATIONS.—There is the same tendency to parenchymatous degenerations of the different organs and tissues of the body in typhus as in typhoid. Usually the body is not very much emaciated; it undergoes decomposition much more rapidly after death from typhus than after death from typhoid fever. In severe cases decomposition apparently commences before death. The muscles are usually of a brownish color, dry, presenting an infiltration of fine granules in the primitive fibres; sometimes hemorrhages take place into them.

The liver and spleen undergo degenerative changes similar to those described as occurring in typhoid, but they are not so extensive nor are they so constant. You may make very many autopsies on persons dying of typhus fever,

without finding any softening or only a very moderate softening of the spleen. The parenchymatous changes in the kidneys are much more extensive and constant in typhus than in typhoid. In severe cases the cortical portion of the organs is swollen, opaque, and more or less fatty, according to the duration and severity of the disease. The primary enlargement of the kidneys is mainly due to a cloudy swelling of the epithelium of the renal tubes.

This tendency to cloudy swelling and granular fatty degeneration, which occurs in the voluntary muscles and the kidneys, also occurs in the muscular tissues of the heart. If the fever is protracted, the cardiac walls become flaccid, of a brownish color, and parenchymatous changes are found similar to those which occur in typhoid fever, though less marked. There is often a considerable amount of serum in the pericardium. Pultaceous clots are found in the heart cavities, and thrombi are found firmly adherent to the walls of the larger veins.

There is the same tendency to ulceration of the mucous membrane of the mouth and larynx as in typhoid fever. In typhus fever the ulcers are deeper, involving more extensively the submucous tissue.

Splenization of the lungs also occurs in typhus as in typhoid fever.

Thus far we have only noticed those lesions which occur both in typhus and in typhoid fever. We now come to those which are found only in typhus.

BRAIN.—Although there is nothing in the appearance of the brain which is characteristic of this fever, yet it is very unlike that met with in typhoid fever. In the latter disease it usually presents an anæmic appearance. In all cases of typhus the cerebral vessels will be found more or less congested.

In some epidemics you will find all the sinuses and blood-vessels of the brain engorged with dark blood, so that when the calvaria is removed the vessels will stand out upon the surface of the brain. In other epidemics, instead of finding intense congestion, there will be a more or less extensive serous effusion into the meshes of the pia mater; the quan-

tity of the effusion varies from one to eight or ten ounces, and it is most abundant upon the convex surface of the brain, although it also takes place to a limited extent into the ventricles. Wherever there is a large amount of fluid effusion there will be little cerebral congestion. The fluid effusion is usually clear; it may be turbid, and when it is so you may be certain that the fever is complicated by meningitis. The arachnoid loses its natural, glistening appearance, and in many instances you will find the membrane dotted over with yellow or yellowish-white spots.

The brain undergoes little or no change unless the fluid effusion is abundant, when by its pressure the sulci are deepened and the convolutions are sharpened.

It will be seen that instead of having little or no serous effusion in the cranial cavity, as is the case in typhoid fever, there is either an intense congestion of the cerebral vessels, or an abundant fluid effusion underneath the arachnoid and into the cavities of the ventricles. In this regard there is a marked difference in the appearance of the brain in these two forms of fever.

ABDOMINAL LESIONS.—In typhus and typhoid fever, the lesions found in the abdominal cavity widely differ. The real pathological distinction is in the presence or absence of intestinal changes. These are present in typhoid and absent in typhus.

In typhus fever there are no changes which show a tendency to ulceration of the intestinal glands, except those which are produced by congestion, such as is frequently seen in scarlet fever and measles, that is, the Peyerian patches present the shaven-beard appearance; while in typhoid fever, either ulceration of the intestinal glands will be present, or the glands will present the appearance which just precedes ulceration. At the post-mortem examination, if ulceration of the agminated and solitary glands is found, we may be certain the patient died of typhoid fever. In typhus fever there is no enlargement of the mesenteric glands, which in typhoid fever is usually present.

The presence or absence of intestinal changes settles the question, is the fever typhus or typhoid?

Complications.—Although the complications which occur in the course of typhus fever are in no way peculiar to it, yet they are of such frequent occurrence, and are developed during its active progress and modify its phenomena to such a degree, that it is necessary that they should be taken into account in the study of its pathological lesions. You will rarely make a post-mortem upon one who has died from this disease without finding the evidence of some complication that has occurred during the progress of the fever. These complications will vary according to the peculiar type of the epidemic which is prevailing at the time the death occurred. In one epidemic the complications will be pulmonary, in another they will be almost exclusively cerebral and spinal, in another nearly all will be glandular in character.

The pulmonary complications are bronchitis, pneumonia, pleurisy, pulmonary congestion, and œdema. In most cases these pulmonary complications are developed during the primary fever, before convalescence commences.

Their advent is always insidious. You may have an extensive capillary bronchitis develop with very few of the rational symptoms of bronchitis present until within a very short time previous to the death of the patient; in fact, the bronchitis might pass unrecognized but for the presence of its physical signs.

All the rational symptoms of pneumonia may also be absent, and still a physical examination of the chest may reveal a whole lung in a state of pneumonic consolidation. The pneumonia which complicates typhus is of the catarrhal variety. It often leads to pulmonary gangrene, so that gangrene of the lung in connection with the development of typhus is not of infrequent occurrence.

Pleurisy is of so rare occurrence that it may be passed with the simple statement that it is an occasional complication, its physical signs only revealing its presence.

At most of the autopsies you make of typhus fever patients you will find there has been pulmonary congestion and œdema. In many cases, when it is associated with capillary bronchitis or pneumonia, it is the immediate cause

of death, and great care should be taken in your physical examinations that you may detect its commencing development.

Laryngitis is often associated with the more extensive bronchitis which occurs during the active part of the fever. The only cerebro-spinal complication which is met with in typhus fever is meningeal inflammation.

As I have stated, in a large majority of autopsies of typhus fever you will find serum in the meshes of the pia mater, but that is not a certain sign that meningeal inflammation has existed prior to death. In addition to the sub-arachnoid effusion, there must be an exudation of plastic material; the arachnoid must have lost its shining appearance, and be thicker than normal. When such appearances are found it shows that the case has been complicated by meningitis. The development of delirium and active cerebral symptoms is not positive evidence that the patient is suffering from meningeal complication, for the delirium and cerebral excitement may occur independently of meningitis. It is by the character of the delirium, and by the change in the pulse and the appearance of the pupils, that this complication is recognized.

Glandular Enlargements.—The glandular enlargements and inflammations which occur in the course of typhus fever are peculiar in their character, and are rarely met with in typhoid, and then are not extensive; but in typhus fever the external glands of the body—especially those about the neck, the parotid and sublingual—often become so much enlarged and inflamed as to interfere with deglutition, and not infrequently these glandular enlargements are apparently the immediate cause of death.

The inguinal glands sometimes become so enlarged as to interfere with the return circulation, and, as the consequence of this interference, swelling of the lower extremities may be developed. There is a swelling of the lower extremities which depends upon a different cause. It may occur at the beginning of convalescence; then the limbs will present very nearly the same appearance as that noticeable in the condition called *phlegmasia dolens*. Under such

circumstances you may think the patient has phlebitis. You will recollect that I have stated to you that the voluntary muscles undergo degeneration, and that the same kind of degeneration occurs in the muscular tissue of the heart. When this does occur the walls of the heart become very flabby, and when this change has reached a certain point there is developed a tendency to the formation of clots in the heart cavities, and a slowing of the general circulation. The result of such retarding or obstruction of the return circulation is the formation of thrombi in the superficial veins, which interfere with the return circulation, and a swelling of the lower extremities follows; this closely resembles that which is seen in phlegmasia dolens. With this swelling of the lower extremities, suppuration and cellular inflammation may occur, which often results in the formation of quite extensive abscesses.

It is an established fact that whenever the return circulation is slowed from any cause in any disease where there is great feebleness of heart power, very frequently thrombi form in the veins of the lower extremities. This is often well illustrated in the latter stages of phthisis, when swelling of one or both lower extremities occurs as the result of the formation of venous thrombi in the superficial veins.

Diseases of the organs of the special senses, which so frequently complicate typhoid, rarely occur in typhus fever, and there are no serious or constant complications of the digestive organs.

We have now noticed the more prominent lesions of typhus fever, and although there are none which can be regarded as characteristic, still they widely differ from those of any other form of fever, and more especially from those of typhoid.

ETIOLOGY.—I now pass from the study of the pathological lesions of typhus fever to its etiology. At the present day this fever is regarded as depending upon a specific poison, of whose exact nature we are ignorant. All observers agree that in the majority, if not in all instances, it is the product of *contagion*, and that the contagion only emanates from the bodies of those who are affected with

the fever. More recent German writers state that the typhus poison is a germ which is capable of indefinite reproduction. This is a matter of theory, and not fact, for no one as yet has been able to determine the existence of such germs either by microscopical or chemical research. Careful clinical observation has established this fact beyond a doubt: that there exists a specific typhus poison, which can be communicated from the sick to the healthy, which some declare is never of spontaneous origin, while others maintain that the poison may be generated "*de novo*."

Some have strenuously maintained that it can be developed by overcrowding and filth; others, who have seen the largest number of typhus fever cases during the past ten years, maintain that at least it is very doubtful whether typhus fever is ever of spontaneous origin. It is possible to develop a fever from overcrowding, imperfect ventilation, filth, and a combination of causes belonging to this category, but such an one is a septic fever, and not typhus fever.

Some observers have gone so far as to express the opinion that scarlet fever and typhus are closely allied both in their etiology and morbid anatomy, and that typhus fever is no more likely than scarlet fever to be of spontaneous origin. The results of my investigation of the origin of the epidemic of typhus fever which prevailed in this city from July, 1861 to 1864, have led me to the belief that typhus poison is of endemic origin—in other words, that there are certain endemic centres; that Ireland, Italy, and Russia are the great centres, and that, whenever it occurs in other localities, it has been conveyed from these endemic centres to those localities.

In the month of July, 1861, in one day fourteen cases of typhus fever were admitted to the fever wards of Bellevue Hospital, of which wards I had the charge. Previous to this time, for several years (I think for more than ten years), there had been no case of typhus fever in the wards of the hospital. Immediately I commenced investigations in order to ascertain the origin of the fever in these cases. I found that the fever had its origin in the upper story of a

rear tenement-house in Mulberry Street, in the most filthy portion of the city. The first case was that of a little girl, who had been brought into the house, ten days before she sickened, from a ship which had come from Ireland, and which had cases of typhus fever on board. Two weeks after her illness commenced, her aunt, the only other occupant of the apartments (consisting of a room and dark bedroom), sickened of fever and died. In gradual succession, nearly every family residing in the building took the fever.

Becoming frightened, some of these families moved into other streets, formed the nucleus for the development of the disease in the different localities to which they removed, and it soon became a widespread epidemic. There were two hundred typhus fever patients at one time in the hospital.

These families were as well nourished and lived in as well ventilated apartments as thousands of their class in other parts of the city. The only difference was that typhus poison was brought to them in the person of the little girl, and, on account of their badly ventilated apartments and their utter disregard of all hygienic laws, they furnished a fit soil for the reproduction and spread of that typhus poison, the constant and unrestrained intercourse between the healthy and the sick being the means by which the fever was spread.

I found unmistakable evidence that persons living in healthy localities, simply by visiting friends sick with the fever, contracted the disease.

The histories of those cases which were developed within the limits of the hospital showed that a residence in an atmosphere necessarily more or less tainted with typhus poison is not sufficient to develop the disease, but that it is necessary for the subject of the contagion to have been brought in contact with an infected person, or within the atmosphere immediately impregnated with his exhalations.

The fact that no employee in the hospital, who was only brought in contact with the clothing of fever patients, contracted the disease, as well as the absence of any evidence that the disease was propagated by such clothing, goes far

to prove that typhus fever cannot be propagated by fomites alone. The certainty with which every unprotected person who was brought in personal contact with fever patients contracted the disease, proves the contagious power of the poison.

The distance that typhus poison can be transmitted through the atmosphere (from the manner in which the disease was contracted by some of the house physicians), would seem to be limited. It has been proved by actual experiment that the contagious distance of small-pox, in the open air, does not exceed two and one-half feet, and it would seem that the contagious distance of typhus fever is even less than two and one-half feet.

The question now arises, can this poison be conveyed in the clothing?

During the epidemic to which I have referred, when typhus fever patients were brought into the hospital, their clothing was removed in the reception room, and afterwards washed and packed away in a lower room of the building. Upon a most thorough investigation made at that time, I found that not a single person contracted the disease whose duty it was to wash or pack away the clothing; but every one whose duty it was to carry the fever patients from the reception room to the hospital ward took the fever. Every physician and nurse who had the care of typhus fever patients contracted the disease; those who were on the surgical service escaped. Every clergyman who came to administer spiritual consolation to patients in the fever ward fell a victim to the disease. I have brought forward these facts to show that during this epidemic there was no evidence that the disease was either of spontaneous origin, or that it was transmitted from the sick to the healthy except by direct personal contagion.

Typhus poison is undoubtedly present in the body exhalations and the expired air of typhus fever patients; but it requires a concentration of the poison to render it infectious. Slight exposure is not sufficient; it requires a concentrated poison and a prolonged exposure. The more nu-

merous the typhus fever patients are, the more powerful does the contagion become; yet a single exposure even to such an atmosphere is rarely sufficient to develop the disease in an individual who is in good health at the time of the exposure.

If any of you are so circumstanced as to be obliged to take the medical charge of typhus fever patients, you should make your visits as short as possible, and when you are about to auscultate the chest of a fever patient, take a full inspiration at an open window, and hold your breath while you are listening to the respiratory sounds, never inhale the air from the bed of the patient as you examine the posterior surface of the chest. As a rule, make your visits short to a typhus fever patient, avoid inhaling the exhalations of the body, never make a visit until after eating; if you observe these simple directions, you will in the majority of instances escape contagion.

The length of the period of incubation varies. For the development of the disease, it usually requires about two weeks of exposure, such as comes to one who is around those sick with the fever. Repeatedly have I noticed this fact in my own case. I have never had typhus fever, and have never taken special care to avoid infection. My immunity is probably due to some special constitutional idiosyncrasy. I have noticed that whenever I enter upon a typhus fever service, I do not experience any effects from the exposure to typhus poison until about two weeks has elapsed, then I begin to suffer from a peculiar form of headache which continues for about two weeks; the period before the commencement of the headache corresponds to the period of incubation, and the period of headache to the average duration of the disease.

At the present day, the established belief is that typhus fever attacks an individual but once, and that those who have had typhoid fever are to a certain degree protected from typhus. Of all the typhus fever patients treated in Bellevue Hospital, only three gave histories of having previously had the disease. I recall the case of a man, seriously ill, who was treated in the fever-tents for typhus fever,

had the characteristic eruption, left the fever-tents well, and in three weeks returned with the fever, and was more seriously ill than during his first attack of the disease.

From the facts which I have brought before you, we must reach the following conclusions :

First.—That typhus fever is due to a specific poison.

Second.—That this poison is communicated from the sick to the healthy only by personal contagion—that is, the recipient of the poison must be brought in contact with the personal exhalations of the infected person.

Third.—That where there is free ventilation, personal contagion is confined to narrow limits.

Fourth.—That the evidences of the spontaneous origin of typhus are not conclusive, although there can be no question but that overcrowding and bad ventilation favor its spread and increase its severity.

Fifth.—Typhus poison passes into the body mainly through the respired air. Whether it can be taken into the system in the food and drink is still an unsettled question.

LECTURE XIX.

TYPHUS FEVER.

Symptoms.

I WILL continue the history of typhus fever by giving you an outline of the phenomena which attend its development, and afterwards speak of some of its more prominent symptoms.

Its advent is usually sudden—there are no constant premonitory symptoms. In some cases, for a few days, there may be a feeling of indisposition, perhaps of headache, loss of appetite, and vertigo; but in a large majority of cases it is ushered in by a distinct chill. This differs from the chill of pneumonia or that of malarial fever, in that it is short, sharp, and sudden. It may amount to nothing more than a chilly sensation. Following the chill there is a severe and steadily increasing headache; it is frontal, and increases in intensity from hour to hour. This is accompanied by a more or less severe pain in the back and limbs, especially in the thighs. The headache of typhus is more constant and persistent than that which attends the development of any other fever; usually, after a few days it diminishes in intensity. A sense of extreme prostration very soon follows the ushering-in chill. In some cases the patient is compelled, within twenty-four hours from the commencement of his sickness, to take to his bed from muscular weakness.

This loss of muscular power will sometimes show itself by the unsteady, tottering gait of the patient, and is more marked in the early stage of typhus fever than it is in any other disease. At one time, while I was making my visit in the fever ward, my house physician, who was sickening from typhus fever, staggered and fell by my side from loss of muscular power. He died on the eighth day of the disease.

Within the first twenty-four hours after the chill the temperature may rise as high as 105° F. or 106° F., although at the same time the patient may complain of a chilly feeling, and will draw up to the fire or cover himself with blankets.

It is a peculiarity of this fever that, during the first two or three days, the patient experiences a sensation of coldness, while the thermometer shows the temperature to range at 105° F. or higher. During the first week of the disease the temperature remains at 104° F. or 105° F. There will be morning and evening variations, most marked at noon and midnight ; but these variations follow no regular course, as in typhoid fever. From the eighth to the fourteenth day the temperature is liable to sudden depression. As a rule, the temperature falls between the eighth and fourteenth day. There is, without doubt, a day of crisis in this disease. In typical cases, before the fourteenth day there is a marked decline, and often a sudden fall in temperature. By the beginning of the second week the temperature ranges at its highest. If there is a sudden rise in temperature during the second week, it is almost certain evidence that some complication exists.

At first the tongue is swollen and covered with a white coating. It presents very much such an appearance as is seen in many nervous affections. As the disease progresses, after a day or two it assumes a yellowish brown color, and the coating becomes thicker ; later it becomes dry, dark, and fissured. Nausea is sometimes present, rarely vomiting. The abdomen is free from pain, except over the liver ; the bowels are constipated. Some enlargement of the spleen can usually be detected quite early.

The pulse is accelerated from the very beginning of the

fever, ranging from 100 in the morning to 110 or 120 in the evening; the acceleration is greater in children than in adults.

At the onset of the fever the pulse is full, but it soon becomes soft and compressible, and finally feeble. It is rarely dicrotic. It is only in the severest cases just preceding death that the pulse becomes irregular and intermitting. The face is flushed, the conjunctivæ injected, the expression of countenance is dull and heavy, and as the fever progresses, the cheeks assume a mahogany color. The sleep is disturbed, and when the patient is awake his mind is confused; in very severe cases delirium is very early present.

Between the fifth and eighth, usually on the fifth day of the disease, an eruption makes its appearance upon the surface. It appears first upon the sides of the abdomen, and gradually extends over the whole anterior portion of the body, except the face and hands. It is more marked upon the trunk than on the extremities. At first the eruption consists of dirty pink-colored spots, varying in size from a mere point to three or four lines in diameter. These spots are slightly elevated above the surface, and temporarily disappear on firm pressure.

After a day or two the eruption becomes darker in color, and assumes a purplish hue. It is no longer elevated above the surface, does not entirely disappear on firm pressure, and the spots have no well-defined margin. This eruption is made up of irregular spots, varying from a point to two or three lines in diameter, either isolated or grouped together in patches, presenting a very irregular outline; in children it often resembles the eruption of measles. When the eruption is abundant it imparts to the skin a mottled aspect, which has given rise to the term "mulberry rash of typhus." Another distinctive peculiarity is, that each spot or patch remains visible from its first appearance until convalescence is established or death occurs, and it is often seen upon the bodies of those who have died of typhus fever.

In some cases of typhus there are only a few spots of the eruption, while in other cases they are very abundant, and

the surface of the body presents the well-marked mottled appearance. In a certain proportion of cases, after the eruption which I have just described has been visible for a few days, there will appear, scattered over the surface, small dark spots, due to minute subcutaneous hemorrhagic extravasation; these are called *petechiæ*. On this account the disease has been called *petechial typhus*; but these *petechiæ* are by no means distinctive of typhus, for they are also met with in other diseases. The majority of cases of typhus which you meet will have no eruption except the "mulberry rash." When the *petechial* spots are present you will find a more severe form of the disease, and more extensive blood-changes than usual.

In all severe cases, at the close of the first week the headache, which has been the most troublesome symptom, disappears, and delirium comes on. The delirium will vary in character and severity in different epidemics, being much more violent and active in some than in others. Sometimes, at the very outset of the disease, the delirium is very active, the patient shouts and talks more or less incoherently, and is more or less violent. If not restrained, he may throw himself out of the window. This period of intense nervous excitement may last two or three days, during which the countenance becomes livid, the conjunctivæ injected, the hands tremulous, and suddenly the patient may pass into a state of apparent coma. It is not that of complete coma, for the patient can be easily aroused; but he lies upon his back, with a tendency to slip down in bed, picking at the bed-clothes. It is not a state of unconsciousness, although one of apparent coma, for the mental processes are going on with great activity, and the imagination will conjure up a great variety of horrid fancies, and the visions which pass before the patient will be distinctly remembered after recovery has taken place. This condition has been called "*coma vigil*." During this period the experience of years may be crowded into a day or an hour, and the patient may feel that he has lived a lifetime while in this state. Those who have the greatest mental power and possess the highest culture have the most distressing fancies

during this somnolent period. If, in this condition, there is a tendency towards a fatal issue, the patient will pass into a more complete stupor, and the coma will become more and more profound; the respiration becomes less and less frequent; the pulse, which has ranged at about 120 per minute, rises to 140 or 150, and finally becomes imperceptible at the wrist; the tongue, rolled into a round mass, becomes brown and dry, so that the patient is unable to protrude it from the mouth; sordes collect upon the teeth; the conjunctivæ are red, and the eyes when open present a leaden appearance. The patient has no longer power to move his body; he lies on his back with his head thrown back, perhaps is only able to make slight tremulous motions with his hands. The urine collects in the bladder, and, if not removed with a catheter, dribbles away. The extremities become cold, but the body temperature remains at 105° F., or it may rise as high as 107° F. or 108° F. In one case under my observation it rose to 110° F. just preceding death, while the extremities were cold.

If the case is tending to a favorable termination, about the fourteenth day of the fever there is an amelioration of all the symptoms. The patient falls into a quiet sleep, from which he awakes conscious and convalescing. The pulse and temperature fall, the tongue becomes clean and moist, the delirium subsides, and there is a desire for food. After two or three days the pulse reaches its normal standard and strength gradually returns. This is an outline of the progress of the disease in a severe case of typhus fever, terminating either in death or in recovery. In a mild case there will be no delirium. The temperature may not rise above 102° F.; the tongue is neither brown nor dry. There is no great acceleration of the pulse, never beating faster than 120 per minute, and that only for a very short period.

During the entire course of a severe or mild case of typhus fever, there is no gastric or intestinal disturbance, no diarrhœa, no distention of the abdomen, no pain in the right iliac fossæ, no gurgling—in a word, no abdominal symptoms.

In mild cases the eruption is never very abundant, but it

appears on the fifth day, and remains visible until convalescence is established.

I will now speak in detail of the more important symptoms ; those, in fact, which determine the character of the fever.

I have already stated that symptoms indicating disturbance of the nervous system are among the earliest and most prominent.

Of these, *headache* is one of the most constant. For the first week or ten days it is severe and persistent, after which time it gradually abates, and disappears towards the close of the second week.

Delirium comes on usually about the eighth day ; sometimes it is present at the onset of the disease. At whatever period it may be developed, it will continue until the termination of the disease. At first the delirium shows itself at intervals during the night, or lasts all night, to disappear during the day. Its character varies from a low, muttering form, to a very active and noisy delirium.

Every possible variation, as it were, is met with during an epidemic of typhus fever. Acute delirium is more liable to be present with the intelligent and highly cultured, while the delirium is usually low and muttering in character in the case of the aged or uncultured.

Stupor or *somnolence* in some degree is seldom absent. It may develop with or without previous delirium. Usually, as the case progresses towards a fatal termination, stupor comes on ; this becomes more and more profound as the disease advances. The patient often lies for hours apparently unconscious, with his eyes open as though awake, but he is absolutely indifferent to all that is going on around him. This is a condition to which the term "coma vigil" has been applied. It is almost invariably followed by a fatal termination. Sometimes coma comes on suddenly, without any antecedent somnolence ; under such circumstances the urine will be found loaded with albumen.

The brain symptoms appear much earlier in typhus than in typhoid fever. *Loss of muscular strength* is an early and striking symptom in typhus fever. In the majority of

cases, it is present from the very first day of the fever. In many cases, as the fever progresses, the loss of muscular power is so great that the patient is unable to turn in bed; the prostration always increases as the disease advances. In some cases there is little loss of strength during the first week, but the prostration comes on suddenly during the second week of the disease. In addition to the general loss of muscular power, in certain cases there is paralysis of some muscles, such as the sphincter ani and the muscles of the bladder, so that the urine and feces are discharged involuntarily. If the muscular coat of the bladder becomes paralyzed, there is retention of urine.

Dysphagia, partial or complete aphonia, and inability to protrude the tongue, are due to paralysis of the muscles.

Muscular tremor is an indication of very great muscular prostration, and is usually met with in the aged and infirm, and in those who have been addicted to the use of intoxicating drinks.

Muscular spasms and subsultus tendinum are present to a greater or less degree in all severe cases; the tendons of the wrist are most frequently affected. One form of these spasmodic movements is manifested by the patient's picking or fumbling the bed-clothes; another by obstinate hiccough. All these symptoms must be regarded as of very grave import.

General convulsions are of rare occurrence; but if they do occur, they must be regarded as an alarming symptom, as they are usually caused by uræmia. They are most liable to occur towards the close of the second week of the fever.

Emaciation is never as marked a symptom of typhus as of typhoid. It is rarely present to any great degree before the third week of the fever.

Temperature.—During the first week of typhus fever there are no such marked typical variations in temperature as are met with in typhoid—none that will enable you to make a diagnosis. Usually the temperature rises rapidly from the very onset of the fever, and in cases of average severity attains its maximum on or before the second or

third day of the disease. At this period the evening temperature will range between 103° F. and 106° F.

Before the temperature reaches its maximum, the morning and evening variations are slight. After the temperature has reached its maximum, for several days there will be little change; but at some time, usually between the seventh and tenth day, there will be a slight remission until the twelfth or fourteenth day, when it rapidly falls, in typical cases that terminate in recovery, to its normal standard.

Occasionally an elevation of two or more degrees precedes the fall. This sudden fall about the fourteenth day is peculiar to typhus. A very high range of temperature during the first week is an indication that severe cerebral symptoms will be developed during the second week of the fever. If a very sudden rise in temperature occurs during the second week, it indicates the occurrence of some complication.

A case of typhus fever may terminate fatally, in which the temperature at no time has exceeded 103° F. In all fatal cases, just preceding death there is usually a rise of two or three degrees in temperature. During the first week of convalescence the temperature often remains below the normal standard, especially in the morning.

Pulse.—The pulse in this fever is usually frequent, soft, easily compressed, and often irregular. The heart may partake of the general muscular weakness, so that the first sound may become inaudible.

In the severe cases, during the first week the pulse may reach 120 beats per minute, after which time it increases in frequency and feebleness with the severity of the general symptoms. By the third day it may reach 120 beats per minute, usually in the milder case it does not exceed on that day 100 beats per minute. If during the first week it continues for three consecutive days so frequent as 120 beats per minute, it is an almost certain indication of danger. The higher the temperature, and the more frequent the pulse during the first week, the more severe will be the symptoms during the second week. If during the second

week it becomes small, feeble, and frequent, perhaps beating 140 or 150 per minute, you may regard the case as a very unfavorable one.

In this disease, a favorable change is often marked—first, by a gradual, and finally by a sudden diminishing in the frequency of the pulse. When this is followed by a sudden increase in frequency, you may look for some complication.

During the first week, if the pulse increases in frequency the temperature rises, and if the pulse diminishes in frequency the temperature falls; but, during the second week, the pulse may increase in frequency, and yet the temperature may fall, and the pulse may diminish in frequency and yet the temperature rise.

The pulse is not an infallible guide as to the condition of the heart, for sometimes the pulse is full and distinct while the heart power is very much enfeebled: on the other hand, the cardiac impulse may appear strong and the sounds distinct, and yet the radial pulse may be imperceptible. In most fatal cases, after the first week the radial pulse is imperceptible for several days prior to death. Although in most severe cases of typhus fever there is a rapid pulse, yet a slow pulse does not necessarily indicate a mild attack.

Eruption.—The general character of the eruption of typhus fever has already been described.

I will repeat some statements already made to you. The eruption appears on the sixth or seventh day of the fever. Its appearance is preceded and accompanied by a fresh redness of the whole surface, on which dark red spots are scattered, giving the skin a mottled appearance. These spots have an irregular outline, and vary in size from a point to three or four lines in diameter. Sometimes they are few in number, but more commonly they are numerous: the larger spots are formed by the coalescence of the smaller ones. At first they have a dusky pink hue, partially or wholly disappearing on pressure, and as the finger passes over them they seem to be slightly elevated. After a day or two they assume somewhat of a brick-dust color, and are but slightly changed by pressure; then the color of the

spots becomes still darker and darker in hue, and finally they are not affected by firm pressure. Another peculiarity is that each patch or cluster remains visible from its first appearance until the termination of the disease. The eruption may appear upon any portion of the body. Usually it first makes its appearance upon the trunk, soon spreading to the extremities; very rarely is it seen on the face. When the eruption is scanty, it is limited to the chest and abdomen. In some patients the eruption, though well developed, is not prominently marked; the spots are pale and undefined, and though grouped in patches are so irregular that they give to the entire surface a faint, dingy appearance. The question now arises, is the presence of this eruption so constant in typhus fever that by it we may with certainty make the diagnosis of this disease?

I believe that it may be discovered by a careful examination in nearly every case of typhus fever; it is more likely to be indistinct in children than in adults.

When typhus fever is prevailing, an ephemeral fever is often met with, which has many of the prominent symptoms, but not the characteristic eruption of typhus fever. This ephemeral fever or febricula is undoubtedly due to typhus poisoning, yet it is not typhus fever. In a case of fever, where there is a question as regards diagnosis between typhus, typhoid, malarial, and septic fever, all of which have many phenomena in common, I should not be willing to make the diagnosis of typhus fever unless the eruption was present.

Respiration.—Usually, during the first week, the respirations do not exceed twenty or thirty per minute, but during the second week they often run up to forty or fifty per minute. In cases where there is great prostration accompanied by stupor, the respirations sometimes fall to eight or ten per minute. Under such circumstances they are often irregular and puffing in character. Hypostatic congestion of the lungs, if extensive, is attended by great frequency of respiration and evidences of cyanosis. The occurrence of these changes in respiration ought always to lead you to make a careful examination of the chest. The

breath of a typhus fever patient has an odor which closely resembles that exhaled by the skin.

The digestive system, which is so greatly affected in typhoid fever, is very little, if at all, disturbed in typhus fever. Nausea and vomiting are rare, and an examination of the abdomen presents nothing abnormal. There is no tympanitis or tenderness on pressure. Spontaneous diarrhoea is of exceedingly rare occurrence; the bowels are generally constipated. Intestinal hemorrhage is of rare occurrence, and when it is present depends either upon congestion of the mucous membrane of the colon or on hemorrhoids, which accompany an engorged portal circulation.

Urine. — The urine in typhus undergoes important changes. The quantity varies somewhat with the amount of fluid taken into the stomach; usually it is diminished during the first week, sometimes to one-fourth the normal quantity. In the advanced stage of severe cases there is sometimes complete suppression of urine, but more frequently the quantity of urine increases during the later stages of the fever.

The quantity of urea excreted in twenty-four hours during the first few days of the fever is increased, and the increase is in proportion to the intensity of the fever. In the majority of cases it remains abnormally increased until the period of crisis is reached (about the fourteenth day), when it gradually, or in some instances rapidly, falls below the normal standard.

In all severe cases, during the first week of the disease, a small amount of albumen is always found in the urine; when the quantity is large, the case may be regarded as very severe.

In the severer cases the urine will also be found to contain vesical and renal epithelium, and when the quantity of albumen is large, epithelial and fatty casts of the uriniferous tubes will be present.

In this connection it is important to bear in mind the necessity of daily inquiry into the expulsive power of the bladder. When there is little cerebral disturbance, the

urine is passed without difficulty ; but when stupor and a tendency to coma exist, there is often retention or an involuntary dribbling of urine, which might lead one to think that there was no accumulation of urine in the bladder.

It is safe to inquire, at least once a day, as to the state of this organ, and if involuntary discharges of urine occur, the contents of the bladder should be evacuated by means of a catheter.

LECTURE XX.

TYPHUS FEVER.

Symptoms.—Differential Diagnosis.—Prognosis.

THIS morning I will speak of the complications of typhus fever, and its differential diagnosis.

In typhus as well as in typhoid fever, you must be prepared for the occurrence of *complications*. Although they do not properly belong to the primary disease, yet they so modify it that they enter very largely into its history. Reference has already been made to them under the head of anatomical lesions, yet it is necessary that I should again speak of them under the head of symptoms. In a large number of cases which terminate fatally, death is due to some one of these complications. Most of these commence before the cessation of the primary fever; occasionally convalescence is interrupted by their occurrence, and indefinitely prolonged. Doubtless, in many instances, they depend upon the weakened condition of the heart induced by the typhus poison. In some epidemics they are all pulmonary; in others they are all cerebral. The advent of pulmonary complications in this fever is always insidious; the cough and expectoration which usually attend pulmonary diseases are either absent, or so slight as not to attract the attention of the physician.

Frequently, rapid breathing and lividity of the face are the first obvious indications of extensive disease of the lungs. When these symptoms are present, a careful physical examination of the chest should be made.

Bronchitis may come on at any period during the fever, and it may continue after the fever has subsided. So long as it is confined to the larger tubes there is little danger, but sometimes suddenly and insidiously it extends into the smaller tubes and is complicated with pulmonary congestion and œdema. Under such circumstances it may be the direct cause of death.

The pneumonia which complicates typhus fever is lobular in character, and frequently is preceded or accompanied by bronchitis. It has a tendency to terminate in abscess or gangrene. During life it is not always possible to distinguish it from hypostatic congestion. If, however, the dullness on percussion is confined to one lung, if the respiration is bronchial and the pneumonic sputa is present, the pneumonia is readily established. The seat of the pneumonia is generally at the upper portion of the lung.

Laryngitis is sometimes a very serious complication of typhus. It may be croupous in character, but the more common form is that of acute œdema glottidis. Its occurrence is readily recognized by the signs of laryngeal obstruction which attend its development. Whenever you meet with extensive swelling of the glands about the neck, with great tumefaction of the mucous membrane of the pharynx, you must be on the watch for the occurrence of this complication.

On account of the extensive blood-changes which sometimes occur in severe cases of typhus fever, the blood readily escapes through the walls of the vessels, giving rise to extensive hemorrhages from the mucous surfaces and into the cellular tissue. The occurrence of the hemorrhages is peculiar to certain epidemics, and when they occur it is during the first week of the fever.

Meningitis is the only cerebral complication which you will probably meet with in this fever. This occurs more frequently in children than in adults, and is not present in every epidemic. The cerebral symptoms, which are such constant attendants upon typhus fever (as I have already stated), do not depend upon meningeal inflammation; they belong to the regular history of the disease. If, during the

course of the fever, there is a deep-seated pain in the head, with restlessness, which shows itself by a constant attempt to get out of bed, with photophobia, contracted pupils, and flushing of the face and eyes, followed by somnolence gradually lapsing into coma, you may be almost certain that meningitis is occurring as a complication. This is most liable to occur during the second week of the fever. The characteristic symptom which marks its development is the constant attempt on the part of the patient to get out of bed. He is so persistent in this that unless watched with the greatest care he will be found upon the floor, vainly attempting to rise. The patient has more muscular power than before the occurrence of the meningeal complication, for he will perform acts which previously he was wholly unable to perform. Usually the delirium lasts two days, then the patient gradually passes into a state of coma from which he cannot be aroused: his respirations may not be more than eight or ten per minute. Dilatation of the pupils, and an intermitting and almost imperceptible pulse, immediately precede death.

I have already referred to the anatomical changes in the kidneys, which are so frequently met with in the course of this fever. I prefer to regard most of these changes as a part of the history of the fever rather than as a complication, although in some few instances croupous nephritis occurs, which must be included in the list of complications. Its occurrence in the course of typhus fever is indicated by the almost entire suppression of urine, and by the presence of albumen in the urine, and exudative and blood casts.

Glandular swellings are also occasional complications of typhus fever, and sometimes may be of a very serious nature, for they may so interfere with deglutition and respiration as to destroy the life of the patient. These swellings usually appear immediately after the crisis of the primary fever. They often enlarge with great rapidity, and in some instances terminate in extensive suppuration.

I have now briefly given you an outline of the symptoms which mark the development and progress of a case of typhus fever, and also of the prominent complications which

may occur during its progress. There are certain accidental or occasional complications which cannot strictly be regarded as a part of the history of this fever, as they may occur with any other fever. To these I shall not refer.

DURATION.—The duration of typhus fever is considerably shorter than that of typhoid fever, and it is of great importance, both as regards prognosis and treatment, to be able to fix the time of its continuance. Usually the day of crisis is between the tenth and sixteenth day. The average duration of the fever is thirteen or fourteen days. It is of shorter duration with the young than with the old, with children than with adults.

Relapses are extremely rare in this fever. I have met with a second and third attack of the fever in the same individual, but I have never met with a true relapse.

Typhus fever varies very slightly in its general character in different cases. Authors have described a number of different varieties, depending on the mildness or severity of the disease, the prominence of certain symptoms, the presence of complications and the circumstances under which the fever appears; but the general description of the fever which I have already given you includes that of the so-called different varieties.

DIFFERENTIAL DIAGNOSIS.—Before the appearance of the eruption, the diagnosis of typhus fever is always difficult, and sometimes impossible. The diseases with which it is most liable to be confounded are *typhoid fever*, *relapsing fever*, *measles*, *pneumonia*, *acute Bright's disease*, *meningitis*, *delirium tremens*, and some of the other acute blood diseases, such as *erysipelas*, *pyæmia*, *septicæmia*, etc.

The early characteristic symptoms of typhus fever are chilliness, pain in the back and limbs, and headache. During the first week the headache increases in severity from hour to hour, and is accompanied by a rapid rise in temperature. These symptoms occurring in one who has been exposed to typhus poison are almost sufficient for a diagnosis. The appearance of the eruption settles the question.

On account of the similarity in appearance of the eruption of typhus fever and that of measles, in children, the one

disease is sometimes mistaken for the other. In both diseases the eruption may appear on the fifth day, but the eruption of measles is of a brighter tint than that of typhus fever, and its appearance is preceded by a cough and coryza, which are not present in typhus fever.

Meningitis.—The differential diagnosis between typhus fever and cerebro-spinal meningitis is difficult. Not unfrequently, days may elapse before you are able to decide whether a case is one of typhus fever or of cerebro-spinal meningitis. To show how difficult is the diagnosis between these two affections, I will mention a circumstance which occurred a short time since in Bellevue Hospital. A patient was brought into the hospital directly from a ship, and the diagnosis of cerebro-spinal meningitis was made by several of the attending staff; but at the autopsy there were found none of the lesions of meningitis, but all the changes corresponded to those found at the autopsies of patients dying of typhus fever.

Yet there are many distinguishing points of difference between the two diseases. The headache of meningitis, at the outset of the disease, is more distressing than that of typhus, and it alternates with delirium. These are the early symptoms of meningitis. When delirium comes on in typhus fever, the pain in the head ceases.

Photophobia and contracted pupils are among the early symptoms of meningitis, and the patient is greatly disturbed by noise, while in typhus fever he seems indifferent to both. Inequality of the pupils, strabismus, ptosis, and paralysis are common in meningitis and rare in typhus. In meningitis the countenance is expressive of pain, wildness, and anxiety; in typhus fever it is blank and stupid.

Again, in meningitis the pulse is first slow and full, then rapid and irregular, and lastly intermitting; while in typhus fever it is rapid at the outset of the disease, and is easily compressed.

Lastly, the eruption of typhus fever is characteristic. If an eruption is present in meningitis, it has no regularity in its development; it may appear within twenty-four hours after the development of the first symptom of the

disease, or it may be postponed for several days, or it may not appear at all. It does not appear on the fifth or sixth day of the disease, with the uniform regularity of the eruption of typhus fever. You may find petechiæ in meningitis as well as in typhus fever, but, as I have already told you, they are not characteristic of the latter disease.

The temperature rises more rapidly in typhus fever than in meningitis, and reaches a higher range. Rigidity of the muscles of the neck is not always positive evidence of meningitis, for sometimes it occurs in typhus fever.

Pneumonia.—Sometimes a latent pneumonia with typhoid symptoms is mistaken for typhus fever; especially is this the case when the latter is prevailing. I frequently saw cases where such a mistake had been made, while in charge of the typhus fever patients on Blackwell's Island, during the epidemic to which reference has been made. In these cases you will have active typhoid symptoms, such as dry tongue, delirium, high temperature, etc. The countenance in this pneumonia, although the cheeks may have a purplish hue, does not exhibit that dull, heavy expression so commonly seen in typhus fever. Although there may be delirium in both instances, the delirium in the former disease is of a milder type than in the latter. The characteristic pneumonic expectoration is not usually present in these cases, and you must not therefore rely upon that symptom in making your differential diagnosis. The physical signs of pulmonic consolidation will lead you to pneumonia, and, unless the typhus eruption is present, this will be sufficient for a diagnosis. If pulmonary consolidation is a complication of typhus fever, it will not be developed until after the sixth day of the fever, the time when the eruption should have appeared. If no eruption is present, the pneumonic consolidation may be regarded as the primary affection, and the symptoms which simulated those of typhus fever may be regarded as secondary.

Delirium Tremens.—The delirium of "delirium tremens" may sometimes so closely resemble that of typhus fever, that the one may be mistaken for the other. The mistake has been made in Bellevue Hospital, and typhus fever pa-

tients have been placed in the cells, supposing them to be cases of delirium tremens. If the "delirium tremens" is uncomplicated by pneumonia, take the temperature of the patient; then it will be very easy to make a differential diagnosis, for in "delirium tremens" the temperature is rarely above 100° F., while in typhus fever with delirium the thermometrical range is 104° F. or 105° F. You may have a rapid pulse in delirium tremens, and often the patient has a brown, dry tongue, and other typhoid symptoms; but there is only a slight rise in temperature; besides, there is no eruption present. The attack is not ushered in by headache, but by an inability to sleep, and the circumstances which precede and give rise to such an attack will establish beyond a doubt the true nature of the attack.

Acute Bright's Disease.—It is not surprising that acute uræmia from acute parenchymatous nephritis should be mistaken for typhus fever. The brown, dry tongue, the tendency to stupor, the contracted pupil, the low muttering delirium, and all the phenomena of the typhoid state, as well as the albuminous urine, belong to both diseases; but the temperature is not raised in uræmia as it is in typhus fever, and the œdema which is always present in acute uræmia is absent in typhus fever.

Erysipelas, pyæmia, septicæmia, and all similar acute blood diseases are often attended by many of the symptoms which attend the development of typhus fever.

In pyæmia and septicæmia you have irregular chills, followed by fever and profuse sweats, with evidences of septic and pyæmic poisoning; in erysipelas, you have the evidences of a localized phlegmon. You must remember that erysipelas is sometimes ushered in by all the phenomena that attend the ushering in of typhus fever; this is before the local inflammation shows itself. In such cases it is impossible to make a differential diagnosis until the local phenomena which characterize erysipelas show themselves, or until the typhus eruption appears. In many of the acute infectious diseases you will be compelled to wait until the time for the appearance of the eruption before you can exclude typhus fever.

When typhus fever is prevailing, and you are watchful in regard to its appearance, you will usually have little difficulty in diagnosis.

You must alway bear in mind that sometimes typhoid, typhus, and relapsing fever prevail at the same time, in the same locality.

The importance of early forming a correct differential diagnosis between typhus and typhoid fever cannot be over-estimated; and in order that you may be the better able to accomplish this, I will now review the prominent symptoms of each, and compare them. By so doing, we shall review their etiology, morbid anatomy, etc.

The *first* point to be considered in the differential diagnosis of these two diseases is, that typhus fever is sudden in its advent, while typhoid fever comes on insidiously, and is slowly developed. In the majority of cases of the former disease there is a chill at the commencement, and severe pain in the head, whereas in the latter there is only a chilliness, some aching in the limbs, and a slight headache. Muscular prostration and progressive muscular weakness appear earlier and are much more marked in typhus than in typhoid.

Second.—The range of temperature in the two forms of fever greatly differs. For example, in typhoid fever we commence on the first day with a slight rise in temperature, which continues, with morning remissions and evening exacerbations, until the end of the first week, when it has reached its highest point; during the second week it remains at about the same degree, with only slight variations; during the third week there are more marked morning remissions; and by the end of the fourth week the temperature has reached its normal standard.

In typhus fever, the temperature rises rapidly, and before the end of the second day reaches 104° F. or 105° F. Whatever degree is reached on the third day may be regarded as the maximum temperature; after this time there are slight, irregular variations until the tenth or twelfth day, when the temperature begins to fall, and rapidly reaches the normal standard.

Third.—These two forms of fever differ very markedly as regards the eruption.

In typhus fever the eruption makes its appearance upon the fifth or sixth day ; while the eruption of typhoid fever makes its appearance between the seventh and ninth day of the fever. The eruption of typhus appears upon the arms and chest, and more or less over the entire body ; whereas the eruption of typhoid appears upon the chest and abdomen, very rarely upon the extremities ; sometimes it appears upon the loins when it cannot be found on any other part of the body. As a rule, the spots in typhus are numerous ; while in typhoid they are not very abundant.

In typhus fever, at first the spots are small, slightly elevated, of a dark pinkish hue, and disappear only on firm pressure. As the disease advances they become darker, and finally are not affected by firm pressure and remain visible from the time of their appearance until death occurs or convalescence is established. In typhoid fever each spot is rose-colored, slightly elevated, and disappears on slight pressure. Each spot remains visible for three days and then disappears, to be followed by another crop. Usually, the eruption is visible about two weeks, and when it disappears leaves the skin unstained, whereas in typhus the eruption disappears and leaves a stain upon the surface. There is a mottling of the surface in typhus fever which is not seen in typhoid, and has been described as the *mulberry rash*.

It would seem as though a differential diagnosis might be as easily made between the eruption of these two forms of fever as between the eruption of measles and that of scarlatina. There may be cases which will cause you to hesitate as regards diagnosis, but when the eruption is developed there need be no question as to which form of fever it belongs.

Fourth.—The brain symptoms in these two diseases also differ. In typhus fever they appear early, and the headache and delirium are more intense than in typhoid. Delirium in typhoid more commonly appears at the end of the second or during the third week of the disease ; whereas in

typhus it appears early, and before the end of the second week has disappeared if recovery is to take place.

Fifth.—As a rule, in typhus fever constipation is present, and you will be obliged to make use of some mild cathartic in order to move the bowels; whereas in typhoid fever diarrhœa is one of the prominent symptoms.

Tympanitic distention of the abdomen, gurgling, and tenderness in the right iliac fossæ, and perhaps intestinal hemorrhage, are all phenomena of typhoid fever, but are never present in typhus fever.

Sixth.—Another point in differential diagnosis relates to the duration of the fever, and here we have a marked difference.

In typhus fever, usually convalescence will be established before the end of the second week; some say the tenth is the critical day, but I think it may be any day between the eighth and fourteenth. The average duration of typhus then may be regarded as fourteen days; whereas in typhoid fever the average duration is from twenty-one to thirty days; twenty-one the minimum, and thirty the maximum number of days.

Seventh.—Typhus fever is contagious; typhoid fever is non-contagious. Typhus fever is due to an animal poison; typhoid fever is due to an animal poison developed in connection with vegetable decomposition.

The fact that one is contagious and the other non-contagious renders the differential diagnosis of great importance.

Eighth.—When we come to the pathological lesions, and consider the manner in which death occurs in these two forms of fever, we readily see how widely they differ.

The characteristic pathological lesions of typhoid fever are the changes which take place in the intestinal glands, such as ulceration or tendency to ulceration. In all cases these characteristic lesions are present.

Suppose you have a case of what you have called typhoid fever, and you follow it to the dead-house, but do not find ulceration or evidences of a tendency to ulceration of Peyer's patches, then you may be quite sure that you have made a mistake in diagnosis.

If, on the other hand, you have a case of supposed typhus fever, and you follow it to the dead-house, and find ulceration of Peyer's patches, you may be equally certain that you have made a mistake, and that you have been treating a case of typhoid, and not typhus fever.

The parenchymatous changes which are common to both diseases have already been sufficiently considered.

Lastly.—Typhus fever is generally epidemic; typhoid is always endemic. In regard to the protection which one attack of typhus fever furnishes against a second attack, it very markedly differs from typhoid fever. One may have typhoid fever whenever the system has been exposed to the typhoid poison; but one attack of typhus is almost a certain protection against a second attack.

Prognosis.—The prognosis in this disease is always grave, and should not be given until you have very carefully considered all the points in each case: such as the age of the patient, the character of the epidemic, and the tendency to certain complications. In all epidemics, the majority of cases will recover. The ratio of mortality as given by different writers, varies from one death in five cases to one death in sixteen cases. The surroundings of each patient should be carefully noted, also the hygienic influences which he is under, and his habits of life should be taken into account. With the intemperate the disease is likely to prove fatal. Some of the circumstances which increase the danger in any particular case are, a debilitated condition of the patient from advanced age, intemperate habits, privation, and previous disease; mental depression, presentiment of death, and over-crowding and bad ventilation: a gouty diathesis is always dangerous. Death may occur in typhus fever from three general causes:

First.—From coma. This is the result of overwhelming the system with typhus poison. The patient does not die from the effect of a prolonged high temperature, nor from complication, but dies as patients die in acute uræmia, because the system is overwhelmed by the typhus poison, and the functions of organic life are arrested by its action on the nerve-centres.

Second.—Death may occur from *syncope* due to heart failure, whether the heart failure is the result of the prolonged high temperature, or the direct action of the typhus poison. A continued temperature of 105° F. or 106° F. is very liable to be followed by fatal syncope from failure of heart power, although the evidences of parenchymatous degeneration of the heart may not be present.

Third.—Death may occur from complication.

Let us now study in detail the individual symptoms and signs which render the prognosis unfavorable.

A *pulse* of more than 120 per minute, continuing a number of days, intermittent, and sometimes irregular, bespeaks an unfavorable prognosis.

A hurried and difficult respiration, with turgidity of the face, due either to cerebral or pulmonary œdema, renders the prognosis unfavorable.

Delirium which is very active and accompanied by great muscular prostration, as indicated by subsultus, slipping down in the bed, and accompanied by that condition known as “coma vigil,” lasting for a number of days, is almost a certain indication of a fatal termination.

The “pin-hole pupil” mentioned by the old writers is an unfavorable omen. It does not necessarily indicate the presence of meningitis, as was once supposed. Great muscular prostration at the very onset of the disease renders the prognosis unfavorable.

Marked impairment of the special senses, accompanied by very great rapidity of the pulse, is an element of unfavorable prognosis.

The more abundant and the darker colored the eruption, especially if accompanied by petechial spots, the more unfavorable the prognosis. In children the eruption is lighter in color than it is in adults, presenting an appearance similar to the typhoid eruption. In adult cases, where there is dark mottling of the surface confined to the extremities, with evidences of blood extravasation, indicated by the presence of petechiæ, your prognosis must be unfavorable, but the case is by no means hopeless.

A dry, brown, retracted, tremulous tongue is seen only in

severe cases. A long-continued high temperature is always an unfavorable symptom. Great diminution in the quantity of urine is an unfavorable symptom, as is also the presence of casts and albumen in the urine. Retention of urine is a more unfavorable symptom than incontinence of urine; convulsions and coma are liable to follow such retention.

You must remember that in typhus fever, more than in any other disease, the patient may pass into an apparently hopeless condition, and afterwards rally and recover. A patient who seems to be overwhelmed with the poison, who has "coma vigil," "pin-hole pupils," rolling of the tongue, and a feeble, irregular, but intermitting pulse, may recover, although these symptoms warrant an unfavorable prognosis.

"Coma vigil," more than any single symptom, warrants an unfavorable prognosis.

The *first indication of recovery* is a diminution in the frequency of the pulse. The pulse may have been 120, but on the tenth, twelfth, or fourteenth day, it begins to diminish in frequency. The tongue has been brown and dry, subsultus and delirium may have been present, even "coma vigil" may have manifested itself; there has been great muscular prostration; the patient, attempting to rise from the bed, may have fallen upon the floor; now, the pulse begins to get slower, the patient falls into a refreshing sleep and awakes perfectly conscious; his countenance is changed from the dusky hue to an almost natural appearance, and he desires food. In other words, within twenty-four hours an entire change comes over the patient, and that change is first indicated by a diminution in the frequency of the pulse, accompanied by a fall in temperature. The fall in temperature is not extreme; perhaps a fall of two degrees is first noticed.

My experience goes to show that there is an attempt at convalescence upon the eighth day of the fever. Especially in those cases that recover, upon that day you will notice a slight fall in temperature, although the temperature may again rise; upon the twelfth or fourteenth day there is a distinct fall in temperature and diminution in the frequency of the pulse that is indicative of convalescence.

The mode of recovery in these two forms of fever, typhus and typhoid, is perhaps the most distinguishing clinical feature. In typhus, recovery is rapid ; while in typhoid it is markedly slow.

Of all the conditions which influence the prognosis in typhus fever, *age* and *the habits of the patient* have as great, if not greater, influence than any other. I am convinced of this from an experience in the care of typhus fever patients which dates back almost to the very commencement of my study of medicine, for very early did I have the care of a typhus fever ward.

In children, typhus fever is a very simple form of disease. The rate of mortality is very low. I remember having the care of sixty children with typhus fever, and among these only one death occurred. This is as low a rate of mortality as you can expect in measles.

When the patient has passed the middle period of life, there is great danger from typhus fever. So with the intemperate, and those who have lived amid unfavorable hygienic surroundings.

The bright, educated person, the one with an active brain, is less likely to recover than is the stupid, uneducated one. For example, the hod-carriers may have the worst type of typhus fever, and pass through it with safety, stupid when they contract the disease, and stupid when they get well. Let a man with an active brain contract the disease, and the "*coma vigil*" comes on, the imagination is vivid ; failure of heart power is present early, and death is almost certain to follow.

LECTURE XXI.

TYPHUS FEVER.

Treatment.

I HAVE already completed the history of typhus fever, with the exception of its treatment, and now invite attention to the more prominent measures which have been and now are employed in its management. You will notice that in many respects these measures are similar to those proposed for the management of typhoid fever patients, yet the treatment of these two diseases differs in certain essential particulars. When the symptoms are mild, very simple measures are all that is required. Of these, confinement to bed, cooling drinks, mild aperients, a milk diet, and free ventilation are the chief, and, indeed, all that is required. It is also important to observe the same rules in regard to the arrangement of the sick-room which were recommended in the case of typhoid fever patients. The more perfect the ventilation, the greater the amount of fresh air around the patient, the better his chances for recovery.

The majority of cases of typhus fever are ushered in by active, and severe symptoms, such as would tempt one to adopt a vigorous plan of treatment—symptoms which at one time were thought to indicate the employment of heroic antiphlogistic measures. You must remember that these active symptoms are due to the effect produced on the nervous system by a poison contained in the circulating blood, and that this cannot be eliminated by any means of which

we have any accurate knowledge, certainly not by vomiting, purging, sweating, or bleeding. With these symptoms there is great prostration of the vital powers and a rapid metamorphosis of tissue. Although the symptoms seem urgent, and the patient has a flushed face, a rapid pulse, congested conjunctivæ, and a high temperature, not a single measure must be resorted to which has a tendency to diminish the vitality of your patient. Dr. Tweede, of London, states, as the summing up of his experience upon this point, that although at one time he supposed bleeding and the so-called antiplhiogistic remedies were necessities in the treatment of typhus fever, yet for the past ten or fifteen years he has not seen a single case in which depletive measures were admissible.

Writers upon this disease usually consider its treatment under two heads—the preventive and curative. I prefer to use the terms prophylactic and remedial or medicinal, for I question our ability to *cure* disease.

You can do much to prevent the development of many diseases, and, as guardians of the public health, this will constitute an important part in the active labor of your profession.

How, then, can you prevent the development of typhus fever? Medical skill cannot prevent the importation of the disease into localities where it is not indigenous. This is beyond the power of medical men, for it is controlled by state and national authority. Consequently typhus fever will probably continue to be imported into districts where it does not originate.

For example, we shall occasionally see the disease in this city; it may appear in any of our commercial seaports, and from them it may be carried into the interior. Yet we can do much to prevent its spread after it is imported, and can prevent its development as an epidemic when it is carried into any locality in the interior. It is important that the first case or cases of typhus fever which are developed in any locality should be closely watched. They should be immediately quarantined. The dwellings in which the fever has broken out should be depopulated, that is, in a tene-

ment-house in which the fever has made its appearance, all the families should be removed, and the house should be thoroughly disinfected. The disinfection must be thorough, not for a few hours, but for one or two days, and afterwards the house should remain open for the free circulation of air for a considerable length of time before persons should be allowed to again inhabit the rooms. Before we conclude the subject of treatment you will see the importance of following these directions.

If typhus fever occurs in the dwellings of the wealthy, their houses must be quarantined. All persons must be prevented from visiting them, and all persons within the dwelling must be prevented from going abroad. After the sick have recovered, there must be the same thorough disinfection as in the tenement house.

All these regulations must be as carefully observed among the rich as among the poor. It is the rule, that though a person may be well fed, well clothed, and well housed, and be ever so cleanly, yet if brought in contact with the poison of typhus fever for a sufficient length of time he will contract the disease.

Usually, in epidemics of typhus fever there are certain foci from which the disease spreads. Perhaps the points from which the contagion more especially emanates are within an area of half a mile square, and yet the disease may have been prevailing for two, three, or even four months. Under such circumstances it is possible to prevent the spread of the fever by the means just indicated.

As far as its management in hospitals is concerned, I would say you should never undertake it within brick or stone enclosures. If possible, patients should be placed in broad pavilions or tents, so that the largest possible amount of fresh air shall be in circulation about them. It is not sufficient to have free ventilation in the ordinary acceptance of that term. The opening of a window will not accomplish the desired result. Remove all the windows in a room, regardless of the cold, and cover the patients with a sufficient number of blankets to keep them warm. Allow fresh air to surround them.

There are certain conditions which predispose to the development of typhus fever, such as the conditions caused by interference with nutrition, by want of cleanliness, bad ventilation, want of food, and habits of intemperance.

In Ireland, when famine occurs, then the people suffer most from typhus fever; then it prevails as an epidemic. When it prevails epidemically in Ireland, then we are almost certain to receive a certain number of cases in New York.

Fatigue, anxiety, and anything which tends to lower the vitality of an individual render him susceptible to the influence of typhus fever poison. Remember this, and also what I have before told you in regard to eating before you enter a ward filled with typhus fever patients.

When the typhus fever manifests itself you can now understand how important it is that the guardians of the poor should not only enforce cleanliness, but that they should feed the poor better than at other times. If cleanliness is observed, the dwellings thoroughly disinfected, and the poor well fed, the most virulent epidemic can soon be stayed. The effects produced by such measures are sometimes wonderful.

In the year 1861, at the commencement of the epidemic, when, as I have before stated, the first case occurred in a tenement-house in one of our down-town streets, it was six weeks before it spread from that locality. The spread of the fever should have been stopped at that point; but very little attention was paid to it, and it began to spread from one point to another, until some six or seven thousand cases were developed. Many of our prominent citizens sickened with the fever and died. This epidemic could have been prevented had measures been taken early to prevent the spread of the disease. It seemed to me that our city authorities were responsible for a large proportion of the deaths which occurred during the prevalence of that epidemic.

We now come to the medicinal treatment of this disease.

MEDICINAL TREATMENT.—As I have already stated, medicines are powerless either to arrest the progress or shorten the duration of this fever.

The first point which I shall discuss under this head relates to neutralizing the poison. This, many authors claim, can be done, and the progress of the disease thus be arrested. In my own experience I have found no medicinal agent which can neutralize or destroy typhus poison, or which has power to arrest the progress or shorten the duration of this fever. Different remedial agents have been proposed for the accomplishment of this result, according to the views held in regard to the nature of the typhus poison, and its effects upon the system.

At one time the mineral acids were supposed to possess this power, and were administered for that purpose; but, at the present time, they have fallen into disuse. The internal use of carbolic acid, chlorine water, creasote, and more recently salicylic acid has been recommended for the same purpose. The inhalation of oxygen gas has also been thought to be of service in arresting the blood-changes, and thus preventing the poison from having its customary effect upon the system. By the stimulation which it produces, the patient may be brought out of an apparent state of coma, and revive in a marked degree; but the relief is only temporary. For a time the patient may improve, his consciousness return, and his appearance indicate that convalescence is established; but his unfavorable symptoms will return, and it will become quite evident that the oxygen has not neutralized the typhus poison.

It seems to me that fresh air is the only thing which has power to neutralize the poison of typhus fever. It certainly possesses this power when external to the body. For example: place a patient sick with typhus fever in a well-ventilated board pavilion, or in a tent where an abundance of fresh air can circulate about him, and it is almost impossible for him to communicate the disease to a healthy person. Again, place a patient in a closed room, perhaps twelve by fourteen feet square, let a healthy person remain with him a single night—probably a much shorter time is sufficient—and the latter will be almost certain to contract the disease. Why is the disease more readily communicated in the one case than in the other? Certainly the

fresh air which circulated about the typhus fever patient must have prevented contagion. Fresh air, when inhaled, produces to a greater or less extent the same effect. You may say, how do we know this? It is known as a clinical fact. I have seen a typhus fever patient, who was apparently overwhelmed by the poison—perhaps within forty-eight hours from the commencement of the attack was in a state of coma, with high temperature, a rapid pulse, etc., and all the symptoms indicating that he was fast succumbing to the disease—when brought from a crowded tenement-house and placed in a tent, where he could inhale plenty of fresh air, within four or five hours from the time of admission begin to rally, and go on to recovery. Fresh air was the only remedial agent employed.

If fresh air does not neutralize the poison, it certainly has some effect in eliminating the poison, and thus mitigating the severity of the fever, and perhaps shortening its duration. If you choose, you may regard it as a remedial agent, for it certainly is of greater value than any so-called remedial agent at our command.

To accomplish the best results, place three or four patients in a tent twenty feet square; the fly of the tent should be thrown up, and if the weather is cold, your patient should be well covered with blankets. By this means you will insure all the advantages of free ventilation.

The question now arises, what therapeutical agents can be employed with advantage, in order to accomplish the desired results? The following are of the greatest importance:

First.—The reduction of temperature.

Second.—The sustaining of heart power.

The former is of as great importance in typhus as in typhoid fever, and the same rules should govern you with regard to the agents to be employed, and the mode of their employment.

As in the management of typhoid, so in the management of this fever, we have two antipyretic agents, namely, the sulphate of quinine and the application of cold to the surface. These agents may be employed separately or in con-

junction. I would here repeat a statement already made, that I believe quinine to be the more powerful antipyretic of the two agents.

You will find that the temperature rises more quickly in typhus than in typhoid, after it has been reduced by the cold bath, and all through the early part of the fever you will be obliged to resort to the bath much more frequently than in typhoid.

The rules for the administration of the baths in typhus fever differ somewhat from those that govern you in typhoid.

In typhus fever, as soon as the temperature of the patient rises to 104° F., he must be placed in a bath the temperature of which is about ten degrees below that of the patient ; gradually, by the addition of ice or ice-water, bring the temperature of the bath down to 68° F. or 70° F. The patient must be kept in the bath until his temperature falls to 101° F. or 102° F., then taken out, quickly dried and placed in bed. For some time after the removal from the bath, the axillary temperature will continue to fall, as the trunk parts with heat to the extremities. As soon as the temperature rises again to 104° F., the patient must receive another bath. If the patient is suffering with intense pain in the head, or is actively delirious during the bath, ice-bags may often be applied to the head with benefit.

If the cold baths do not readily reduce the patient's temperature, or if the fall is of short duration, antipyretic doses of quinine must be administered, according to the rules given for its administration in the treatment of typhoid fever.

As soon as you have passed the first week of the disease, having kept the patient's temperature below 103° F., usually it will not be necessary or advisable to continue the baths. In most cases antipyretic doses of quinine will be found sufficient to keep down the temperature. *Now*, if not before, there will be evidence of heart failure, and the question presents itself, Shall alcoholic stimulants be administered ? In this connection I will mention the rules which have governed the profession in the administration of stimulants in typhus fever.

The history of alcoholic stimulants in the treatment of typhus fever dates back about forty years, to the teachings of Graves and Stokes, since which time until quite recently they have constituted an important element in the treatment of this fever, receiving the approval of almost the entire profession. Even at the present day the habit of administering alcohol in large quantities in fever, and not unfrequently in an injudicious manner, has become almost universal. Most writers have regarded a frequent feeble pulse, with feeble cardiac impulse, even though cerebral symptoms may be present, as certainly indicating the administration of alcoholic stimulants. The directions were, to commence their administration early, and in sufficient quantities to control the pulse. It was thought that the earlier their administration commenced, the better the chance for recovery, as the failure of heart power, which makes its appearance in the later stages of typhus, would be prevented. No limit was given as to the quantity to be administered; and when typhus fever was treated in Bellevue Hospital, not unfrequently it was forty or fifty ounces of whiskey administered in divided doses within twenty-four hours.

The object to be accomplished was control of the pulse. This could in most cases be done for a time, but as the disease advanced, and the patient became more and more overwhelmed by the typhus poison, alcohol lost the power of giving force to the pulse. Under such circumstances, the rule was to give it *ad libitum*, for alcohol was regarded as the only agent by which the life of the patient could be saved. I remember administering from a pint to a quart of brandy to a fever patient within twenty-four hours. Now, what is the effect produced by the administration of large quantities of alcohol into the system?

After carefully studying for two years the action of alcohol on typhus fever patients, I became convinced that in some patients, if not in all those who were severely ill, especially where there was interference with the function of the kidneys, its beneficial effects were doubtful, if its action was not decidedly injurious. That stimulants will control

the pulse and sustain the heart's action for a time, there can be no question; but I found that in all severe cases there came a time when alcohol, in however large doses it was given, ceased to have this power. Besides, it must be remembered that large quantities of alcohol thus administered disturb nutrition, lessen secretion, prevent the elimination of urea, and tend to induce a state of coma which cannot readily be distinguished from that induced by the disease itself; all of which must necessarily greatly increase the danger of a fatal termination.

During the prevalence of the last epidemic of typhus fever, I took charge of the fever-tents on Blackwell's Island, with the intention of testing the effect of the withdrawal of stimulants in the treatment of typhus fever.

In my earlier professional life I was thoroughly imbued with the idea (for I was almost born into the profession from a typhus fever ward) that alcohol was a necessity in the treatment of typhus. My house physician, Dr. Engs, who took the immediate care of the fever-tents under my direction, had had a large experience in the treatment of typhus fever in Bellevue Hospital, had there contracted the disease, and believed that his life had been saved by the free use of stimulants.

As we assumed the charge of the tents I ordered that no stimulants nor medicines should be administered to any inmate of the tents.

The cases, as they were brought into the tents from the city, were of as severe a type as any we had treated in Bellevue Hospital: some were in a state of coma, with an imperceptible radial pulse, and all the signs of speedy dissolution,—conditions which I had been educated to regard as most certainly indicating the free administration of stimulants.

The rule which I established was faithfully carried out with the following results: While the fever was in Bellevue, the ratio of mortality was one death in every five; and in the tents, one in sixteen. I do not claim that the great diminution in the ratio of mortality in the tents, as compared with that of Bellevue Hospital, was due to the non-administration of stimulants in the one case, and their free admin-

istration in the other. I do, however, most certainly affirm that my experiments in the tents convinced me that the beneficial effects which had been ascribed to the use of alcohol in typhus fever were not fairly due to it. Although I would not entirely discard the use of alcohol in the treatment of typhus, still I would greatly limit its use and give it only as an occasional aid, to carry my patient over some peculiar time of danger from heart failure.

Typhus fever patients under twenty-five years of age rarely require or are benefited by alcohol, unless they were of intemperate habits prior to the attack. To the old and feeble its occasional administration may be of great benefit, and at times be the means of saving life.

A copious dark eruption, with coldness of the extremities, specially indicates the use of alcohol.

As a rule, delirium, headache, scanty urine, and intense heat of surface contra-indicate the use of alcohol.

In any case when you decide to administer alcohol, carefully watch the effect of the first few doses; the same rules should govern you that were laid down for the administration of stimulants in typhoid fever. It is impossible to give any positive instructions as regards the quantity of stimulants required in each case. It is very rarely necessary at any time during the fever to give more than eight ounces of brandy during twenty-four hours. If this amount will not sustain the heart power, I am confident larger quantities will fail to do it, and also that such administration has hastened the fatal issue.

As soon as the symptoms, on account of which the alcohol may have been resorted to, are relieved, the quantity must be reduced, or its administration altogether stopped. I do not altogether condemn the use of stimulants in typhus fever, but I do so as regards stimulants as a plan of treatment; and, where the patient can be freely exposed to fresh air, I doubt if their use is often required.

To diminish the frequency of the pulse, when it follows the reduction of the temperature by the application of cold to the surface, and the administration of quinine in antipyretic doses, cardiac sedatives have been employed, such

as veratrum, aconite, and digitalis. The rapid pulse in typhus fever, after the first onset of the disease, often is not due to the high temperature, but to the failure of heart power; when such is the case, digitalis should be employed. Digitalis diminishes the frequency of the pulse, by increasing the power of the heart, and at the same time it increases the secretion of urine, which frequently is scanty, and thus, to a limited extent, becomes an eliminative.

From four to six drachms of the infusion of digitalis may often be given with benefit during twenty-four hours. If the heart power cannot be sustained by the moderate use of stimulants and by digitalis given as indicated, we are helpless so far as remedial agents are concerned.

The treatment of the special symptoms of typhus fever require only a passing notice.

The headache, when intense, is best relieved by cold applications in the form of ice-bags. If it is accompanied by intolerance of light, a blister to the back of the neck will be found to give relief.

Sleeplessness in any stage of the disease, if it continues for two or three days, must be relieved, for it is of itself sufficient to cause a fatal termination. If sleep does not follow the applications of cold to the head, opiates may be administered in full doses. I have seen typhus fever patients that had not slept for forty-eight hours drop into a quiet sleep within a few hours after they had been exposed to free ventilation. Great care should be exercised that their apartments are kept perfectly quiet and darkened. When delirium and other cerebral symptoms are associated with sleeplessness, hydrate of chloral may be carefully employed. Stupor is to be counteracted by promoting the action of all the excreting organs, applying external stimulants, and administering diffusible stimulants, the most serviceable of which are coffee, musk, and camphor. In the early stage of the disease the cold douche may be employed.

Two remedies have been recommended for the coma of typhus, namely, valerian and phosphorus; neither of these remedies have seemed to me to be efficacious.

When there are evidences of great prostration in connection with any of these special symptoms to which I have referred, the moderate administration of stimulants may be resorted to, and if relief follows the first few doses their use may be continued.

In the treatment of the complications which I stated to you were liable to occur during the course of typhus fever, you must be guided by general principles and by the symptoms in each individual case, never forgetting that the primary disease has a tendency to induce great nervous prostration and depression, and that the heart's action forbids the use of all depleting remedies, and indicates a supporting plan of treatment.

The pulmonary and laryngeal complications, as well as erysipelas, bed-sores, and gangrene, are to be managed in the same manner as was proposed when they occur as complications in typhoid fever.

Diet.—This is of primary importance. Though the patient refuse all nourishment, if possible he must be required or even compelled to take it. As the digestive powers are impaired, great care is required in selecting and administering the proper nourishment, and it must be given at stated intervals, varying from one to two hours. Care must be taken not to over-feed—much harm may be done in this way. When the patient clinches his teeth and obstinately refuses all food, or is unable to swallow, his life may sometimes be saved by pouring liquid nourishment into the stomach by means of a long tube passed through the nose.

Milk best serves the purpose as an article of diet. It may be given ice-cold, if desired, and in such quantities as the stomach can receive and digest. If more concentrated nutrition is desirable, the yolk of eggs may be beaten up and added to the milk.

The management of patients during convalescence from typhus fever is a matter of very great importance.

As soon as the fever ceases, most patients convalesce rapidly unless there is some complication, and the chief duty of the physician is to prevent premature exertion and exposure to cold, and to restrain the patient in the gratifica-

tion of an inordinate appetite. At this time porter or ale may be taken to increase the power of assimilation. The mineral acids, Peruvian bark, and iron may also be given as tonics; these are particularly called for when the pulse is slow and feeble.

It is important to guard against any sudden physical effort during the early period of convalescence, as it may lead to coagulation of blood in the veins. An opiate or hydrate of chloral is sometimes required to produce sleep during convalescence.

In all cases great benefit will be derived from a temporary change of residence, and daily exercise in the open air.

LECTURE XXII.

RELAPSING FEVER.

*Morbid Anatomy.—Etiology.—Symptoms.—Differential
Diagnosis.—Treatment.*

HAVING completed the history of typhus fever, I shall this morning invite your attention to the next in the list of contagious fevers, namely, *relapsing fever*.

This is no new form of disease. It was described more than a century ago by Dr. Ratty, and since that time has prevailed as an epidemic disease in most of the countries in the northern part of Europe. There is no reliable history of its occurrence as an epidemic in this country until about four years ago, when an epidemic prevailed in this city. It has been reported that in the year 1844 a vessel landed, in Philadelphia, passengers ill of relapsing fever. At one time, while typhus fever was prevailing in Buffalo, some twelve or fourteen cases of relapsing fever were reported, but it is altogether probable that they were cases of irregular typhus fever, for when relapsing fever has been introduced into a locality it is not limited to one or two dozen cases.

MORBID ANATOMY.—In this disease there are no pathological lesions of so uniform occurrence as to indicate its special anatomical character. In a word, there are no characteristic lesions. There are changes present in some of the organs which very closely resemble those that are met with in typhus.

Spleen.—In the majority of autopsies, if death has oc-

curred in the active period of the disease, the spleen will be found considerably increased in size, the capsule thickened, smooth, tense, and slightly clouded, the trabeculae of the organ increased in size, and the Malpighian tufts more prominent than normal. In some cases the spleen will be found enlarged, soft, and flabby. There is no uniform change in its substance, although it is always increased in size during the active period of the disease. After this period has passed it will be found diminished in size, and its surface will present a shrivelled appearance, with the corpuscles rolled into folds. In many cases a number of rounded or irregular miliary masses, of a dull yellow color, will be found.

Liver.—During the active period of the fever this organ will also be found enlarged, and enlargement of the liver is more likely than enlargement of the spleen to remain after this period has passed. The structural change which takes place in the liver is similar to that found in the spleen. The urine often presents a cloudy appearance. The gall bladder is generally distended with dark yellow bile.

Kidneys.—The kidneys will be found increased in size. The increase is due to congestion of the cortical substance, and a granular infiltration of the epithelium of the uriniferous tubules. It is a change similar to that noticed in other fevers.

Intestines.—As a rule, you will find enlargement of the glandular follicles of the intestines. The solitary glands are more commonly affected, but even the Peyerian patches may present the “shaven-beard” appearance. The mesenteric gland may be slightly enlarged, but will not present any change indicative of an inflammatory process, although there is some congestion. Its appearance is similar to that noticed in typhus and typhoid fevers.

Mucous Membranes.—In the majority of cases you will find small spots of blood-extravasation upon the mucous surfaces, especially the mucous membranes of the stomach and intestines, and they may be found on the mucous membranes of the bronchial tubes. These spots of ecchymosis

are present perhaps as constantly as any pathological lesion of the disease.

Blood.—The blood coagulates imperfectly, as in typhus and typhoid fevers.

The heart presents no constant changes. In some cases fine granular infiltration of the muscular fibres has been observed. This same granular infiltration is also sometimes seen in the voluntary muscles.

All the other changes found are those which come under the head of complications.

ETIOLOGY.—There have been wide differences of opinion and much discussion in regard to the etiology of this disease.

At the present time it seems to be the unanimous opinion of those who have had the best opportunities for study, that it is a contagious disease, and that it is a distinct type of fever. Although it presents many phenomena which ally it to typhus, and many other phenomena which ally it to malarial fever, it is neither typhus nor malarial, but is a distinct type of fever having a distinct poison. From observations which have been made upon the blood of patients suffering from this fever, distinct organisms which have the power of developing the fever are thought to have been found.

Several German observers, Cohnheim and others, have given drawings of these organisms, which seem to be little spiral lines that are constantly in motion, and these observers tell us that they are distinctive of this form of disease, and are always present during its active period. They are absent in the interval between the primary attack and the relapse, but are to be seen as soon as the relapse occurs. With reference to these animal organisms, and others which are claimed to be the cause of fevers and other infectious diseases, while it may be true that distinct forms are found in different forms of fever, I question very much if by the introduction of these organisms into the system the fever can be developed. In relapsing fever, more than in any other, have these organisms been seen and studied, and yet all experimenters have failed to develop the fever from

them. This fact gives those who do not believe that living organisms are the cause of infectious diseases a very strong argument; yet, on the other hand, does nothing for those who hold the chemical theory of disease.

It seems of service to those who believe that every disease has its own specific virus, which, as yet, we have not been able to distinguish either by its microscopical outline or by chemical analysis, but which is believed to be a subtle agent, similar in some respects to the venom of animals, and which acts upon the blood in such a manner as to cause the development of the living organism; this organism can be seen under the microscope.

Clinical experience has settled the question, Is relapsing fever a contagious disease, and can it be propagated by personal contagion? Some have maintained that it may be conveyed in the atmosphere, in water, and in clothing. Some of the clinical facts placed on record a few years ago, while the disease prevailed in Germany, go to prove that the fever can be conveyed from the sick to the healthy by means of water; but in Ireland, where the fever seems to be indigenous, there is no such evidence on record. Usually it has prevailed in Ireland when there has been a scarcity of food, and on this account it has been named *famine fever*.

However, the disease is not necessarily accompanied by starvation, for it is developed among those who are well fed as well as among those who are badly nourished. As in typhus fever, there is a connection between the development of an epidemic of this fever and imperfect ventilation and bad hygiene.

I had never seen a case of relapsing fever until about four years ago, when the epidemic prevailed in New York. At that time patients were brought into my wards in Bellevue Hospital with a fever differing from typhus fever by the absence of an eruption, from intermittent in the order of its development, and not closely resembling remittent fever. It seemed to me an irregular form of malarial fever, differing from any form with which I was acquainted, as at that time I was practically unacquainted with the phenomena of relapsing fever.

Eight cases were brought in. From these my house physician contracted the fever, and during his illness I reached the diagnosis of relapsing fever. Subsequently we had large numbers of relapsing fever patients, and a hospital was established for their reception on Hart's Island.

In every case that occurred at that time, where the origin of the fever could be traced, it was found that there had been direct exposure, and it was established beyond doubt that the first cases were brought from Ireland. The contagious character of the affection was also established by the fact that all the nurses and all the physicians who were in immediate attendance upon the sick contracted the fever. If a patient was placed in a bed before it had been cleaned, previously occupied by a person sick with relapsing fever, he was almost certain to contract the disease. At the time of this epidemic we found no evidence that the fever was conveyed by clothing, although some British writers have claimed that it can be done. When our patients were admitted into the hospital, their clothing, as it was removed, was simply washed, not disinfected in any special manner, then packed away, and not a single person who was thus brought in immediate contact with the clothing contracted the disease. The period of incubation ranges between five and seven days.

SYMPTOMS.—The symptoms which usher in relapsing fever are usually well marked. It is sudden in its advent. This is marked by a severe rigor or by a distinct chill. Accompanying the chill there is a frontal headache, pain in the limbs, more or less pain in the back, nausea, and not infrequently vomiting. A rapid rise in temperature follows the chill, and with its appearance the headache increases, as does also the pain in the limbs, especially about the joints. There is vomiting, at first only of the simple contents of the stomach, afterwards of yellowish material. This may be followed by the ejection of a dark-colored material, which very closely resembles the black vomit of yellow fever.

In this disease, the rise in temperature is always rapid, and usually marks its highest point within the first twenty-four hours; during this time it may rise from $98\frac{1}{2}^{\circ}$ F. to

104° F., or even as high as 109° F. From this time, for two or three days, there is usually very little variation. With the occurrence of the chill and fever there is also a rapid increase in the frequency of the pulse. In no disease does the pulse so quickly become rapid as in relapsing fever. It is not uncommon for it to reach 140, 150, or even 160 beats per minute within the first twenty-four hours. It is usually small and compressible.

There is nothing peculiar about the countenance of the patient, but it presents the ordinary appearance noticed in an active febrile excitement.

As the disease progresses the patient becomes more and more prostrated; by the second day he may be unable to turn in bed. The arthritic pains increase in severity, and often become the most distressing symptoms of the fever. As early as the second day, patients begin to complain of a feeling of weight and uneasiness in the upper part of the abdomen, more severe in the left than in the right hypochondrium. Usually there is considerable enlargement and tenderness of the liver. The spleen also becomes rapidly enlarged, and its enlargement is attended with quite severe pain and tenderness. The muscles of the body are, however, the seat of the most severe pain, which is increased by movement and by pressure; the pain is piercing and lancinating in character. On account of this pain, the patient usually lies perfectly quiet; he is not restless, but sleepless. Delirium is not an unfrequent symptom, and is sometimes very active, yet in the majority of moderately severe cases the mind remains undisturbed. There may also be present irregularities of the pupils, photophobia, and other symptoms which might lead you to the diagnosis of meningitis were it not for the character of the pulse.

As the disease progresses, in a certain proportion of cases jaundice is developed; this is usually accompanied by vomiting and severe diarrhoea, and these symptoms seem to ally the disease to some forms of malarial fever.

The great prostration and rapid rise in temperature ally it to typhus fever, but the rise is more rapid and reaches a higher point within the first twenty-four hours than it does

in typhus fever. There is sometimes a slight rose-colored eruption resembling roseola, but having none of the characteristics of typhus eruption. The patient goes on from day to day gradually getting worse, the fever becomes more and more intense; loss of strength and emaciation is progressive, and the muscular pains are more severe. You may have been watching your patient with the greatest anxiety, the pulse has reached 160 per minute, the tongue is brown and dry, extreme nausea and vomiting are present, and the severity of the symptoms indicate that death may speedily occur, when, on the seventh or eighth day of the fever, suddenly a remission occurs, attended by a profuse perspiration. With the occurrence of the profuse sweating the temperature falls; in a few hours it may fall five, six, or even seven degrees; the pulse becomes less frequent; the respirations, which have been hurried and difficult, become regular; the pains in the head and limbs pass away, the thirst disappears, the tongue becomes moist; the engorgement of the liver and spleen rapidly diminishes, as is shown by the rapid diminution in the size of these organs, which is readily determined by percussion.

Within twelve hours from the commencement of the remission, the temperature may fall to less than 100° F., perhaps below the normal standard, and the pulse may fall to 80 or 90 beats per minute.

Sometimes, instead of a profuse perspiration taking place at the commencement of the remission, a profuse hemorrhage from the nose, the bowels, or uterus may occur.

As soon as the remission occurs the patient feels perfectly well, except a sense of weakness. He gets out of bed, and, if he is in a hospital, perhaps insists upon his discharge; his appetite begins to return, and he appears to be rapidly convalescing.

His apparent convalescence is of short duration; sometimes in three or four days, usually at the end of a week, certainly by the twelfth or fourteenth day of the disease, all the phenomena of the primary fever are suddenly developed, or what is termed the relapse occurs. Sometimes

the relapse occurs in the morning, sometimes in the afternoon, but more frequently it comes on at night.

The relapse may be ushered in by a chill, or it may occur without a chill. The pulse may begin to increase in rapidity and in twelve hours reach 140 per minute. With the rapid pulse, the temperature rapidly rises to 160° F. or 170° F. and even as high as 180° F. Usually the fever which attends the relapse is more intense than the primary fever, the liver and spleen become as enlarged as during the primary fever.

It is claimed by some observers that the parasites which are said to be present in the blood during the primary fever, disappear during the remission, but reappear in greater numbers during the relapse. The relapse usually lasts three or four days. In a few cases I have seen it last six or seven days, and in some it does not continue more than forty-eight hours. After it has continued a certain period, a second remission is developed; this, like the first remission, comes on suddenly, is accompanied by a profuse perspiration, and in twenty-four hours from its commencement the pulse and temperature have reached their normal standard. From this period, the patient usually goes on to complete recovery.

As many as three or four relapses may occur, but ordinarily the convalescence becomes complete after the second remission.

Convalescence from relapsing fever is usually rapid, but the patient for a long time remains in a weak condition, suffering more or less from arthritic and muscular pains. The appetite returns slowly. An anemic murmur, which is often very distinct during the active period of the fever, is heard for two or three weeks after the commencement of convalescence. Oedema of the feet, due to general anemia, is often quite marked during convalescence.

The period of convalescence is usually as long as both the period of fever and remission; not unfrequently six or eight weeks elapse before relapsing fever patients are able to resume their accustomed avocations.

At the commencement of convalescence, the decrease in

the size of the spleen is rapid, but frequently it is a long time before the organ reaches its normal size.

COMPLICATIONS.—Few complications have been noticed during the course of relapsing fever. In some epidemics pneumonia has occurred quite frequently; at other times it has been exceedingly rare. When it does occur, it is often double.

Sudden collapse may occur as a complication of relapsing fever, either during the primary fever or during the relapse. The pulse suddenly becomes small, irregular, or intermittent, sometimes imperceptible. The cardiac impulse is feeble, the heart sounds are lost, and the patient rapidly passes into a condition of collapse, and dies. The collapse may come on suddenly in cases previously mild.

Post-febrile ophthalmia is another very remarkable complication or sequela of this fever. It has been observed in most epidemics. It presents two distinct stages, the amaurotic and the inflammatory. During the first stage the patient complains of impaired vision, with motes and luminous circles floating before the eyes. The inflammatory stage is characterized by intense circumorbital pains and lacrymation, without injected conjunctivæ or marked constitutional disturbance. Recovery is tedious, and, unless the case is carefully treated, may end in complete loss of sight. Both eyes are rarely attacked; the right eye is most frequently affected.

Diarrhœa and *dysentery* are common complications, and in some epidemics they are the chief cause of death. They are most likely to come on during the relapse. In our epidemics the most frequent complication is hemorrhage from the mucous surfaces, especially from the stomach and intestines. In two cases that came under my observation hemorrhagic pachymeningitis was the cause of death. In very rare instances, abscess of the spleen, accompanied by pyæmic symptoms, have occurred during the relapse and convalescence.

Pregnant females, no matter at what stage of pregnancy, usually abort during an attack of relapsing fever.

DIFFERENTIAL DIAGNOSIS.—The diagnosis of relapsing

fever is not difficult if you have the entire history of the case; but, at the commencement of an epidemic, during the primary fever, you will necessarily be in doubt as to your diagnosis.

The diseases with which it is possible to confound relapsing fever are typhus, typhoid, remittent, yellow, and dengue fever, small-pox (before the eruption), and measles.

It differs from all these diseases in the suddenness of its invasion, in the short duration of the primary fever, and in its termination in a crisis, and in the almost uniform occurrence of a relapse between the third and fifth days.

Then the muscular and arthritic pains, which are such constant attendants of relapsing fever, distinguish it from the other forms of fever.

A severe form of relapsing fever, attended by jaundice, resembles very closely, in its general appearance, yellow fever; but the high temperature and rapid pulse which attend the development of the former readily distinguish it from the latter; besides, when the relapse comes on, there can no longer be any question as regards diagnosis, for yellow fever is a disease in which a relapse rarely occurs.

Small-pox simulates relapsing fever only during the period of invasion. You need make no doubtful diagnosis after the third day, when the red spots appear along the edges of the hair.

PROGNOSIS.—The prognosis in relapsing fever is always good. During our epidemic about three per cent. of all the cases treated in hospital terminated fatally. This is a lower rate of mortality than we have with measles. Usually deaths from relapsing fever occur, not from the disease, but from some complication. During the epidemic in this city, syncope during relapse was the most frequent cause of death.

Relapsing fever patients may die of hemorrhage from some of the mucous surfaces. A fatal termination may occur from bronchitis, pneumonia, or other pulmonary complications. During one Russian epidemic parenchymatous hemorrhage was a frequent cause of death.

Diarrhœa and dysentery occurring during convalescence sometimes cause a fatal termination.

Sudden suppression of urine dependent upon renal congestion may give rise to acute uræmia, and thus cause death.

My own experience leads me to the belief that the greatest danger in this disease arises from sudden syncope. I remember one very marked case, that of a young physician, who seemed to be doing well in his second relapse, when suddenly he passed into a state of syncope and died. At the post-mortem examination no condition of the internal organs was found which would account for his death.

TREATMENT.—Dr. Reilly, who wrote upon this disease more than a century ago, stated that all those cases of relapsing fever which were abandoned to *whely* and the good providence of God, recovered. The experience of a century has furnished no accepted plan of treatment. The profession are still unsettled as to the best course to be adopted in the management of this disease.

When this fever appeared in our midst, we thought we could control it by large doses of quinine, but we soon found that quinine was of no service in its treatment. Then aconite and veratrum were employed in full doses as antipyretics, but after a time these were abandoned as useless.

Cold baths were resorted to, as also was sponging of the surface in order to reduce the temperature, but in their use we were disappointed. The temperature was reduced while the cold was being applied, but rose again very soon after the patients were removed from the baths, and there was no effort made to diminish the severity or shorten the duration of the primary fever, or prevent the occurrence of the relapse. Opium in full doses was then tried, but with equally unsatisfactory results, although its free use was found to give more comfort to the patients than did any other plan.

In some cases stimulants were administered quite freely, but without any apparent beneficial results.

The conclusion arrived at was, that relapsing fever patients were as well without as with medication. I would insist that relapsing fever patients should be kept quiet in bed during the primary fever, and should not be allowed

to leave their rooms until the period of relapse shall have passed, and that the greatest care should be exercised to guard against the occurrence of syncope. If there is evidence of heart failure, digitalis and stimulants should be administered according to indications. More than this I have nothing to suggest. My experience leads me to place relapsing fever patients under the best hygienic management, with free ventilation and a mild diet, and then carefully watch lest some complication should occur.

LECTURE XXIII.

EXANTHEMATOUS FEVERS.

Small-Pox.—Morbid Anatomy.—Etiology.—Symptoms.

THIS morning I shall commence the history of the exanthematous fevers. They are three in number, *small-pox*, *scarlet fever*, and *measles*, or variola, scarlatina, and rubella. These are distinct diseases, more markedly so than any of the varieties of fever which have been engaging our attention.

Some writers, not regarding them as distinct diseases, have described them under the general head of acute exanthematous diseases. It seems to me that they should be included in the list of fevers: *first*, because they are infectious, and depend for their development upon distinct poisons as specific in their nature as those that develop typhus or typhoid fever; *second*, for the reason that active febrile symptoms attend their development and mark their progress; *third*, because they run a definite course, one marked by regular stages of development and decline, and with rare exceptions they attack the same person but once.

I shall speak first of *small-pox* or *variola*. Since the day of Jenner's wonderful discovery, small-pox has not occupied the attention of the profession as it did previous to that time. Prior to this discovery, small-pox was dreaded like the plague, and when it did prevail, cities, and often whole countries, were depopulated by it. With the discovery of vaccination, a new era was developed in its history. During the present century epidemics of small-pox have

not been greatly feared. During the past six or eight years, however, this disease has been on the increase, and the death records in this city show that some years there have been more deaths from small-pox than from either of the other exanthematous fevers. Why this great increase, is a question worthy of careful consideration. It cannot be from any failure in the protective power of the means which we possess for preventing its development, but from the imperfect manner in which such means are employed. Vaccination, properly performed, is a perfect protection against its development. The truth of this statement no one of extended experience will question, although vaccination, as formerly practised in this city, seemed to fail to protect the masses from the contagion of small-pox.

I shall consider this part of its history more fully under the head of vaccination.

I will now call your attention to the anatomical lesions of small-pox. Besides those which occur upon the mucous membranes and skin, congestions of the internal organs may be regarded as the most prominent. The anatomical changes which occur in all cases will vary in degree, if not in kind, with the type of the variola.

Three distinct types of this disease are recognized, to which have been given the names "*variola discreta*," "*variola confluenta*," and "*variola hemorrhagica*."

MORBID ANATOMY.—You will rarely make a post-mortem examination upon one who has died of small-pox, without finding more or less intense congestion of the lungs, the brain, the liver, the spleen, and the kidneys.

Perhaps the most constant lesions affecting the viscera are parenchymatous degenerations; sometimes these are simply granular infiltrations, at other times they consist of an acute fatty degeneration, resembling that produced by phosphorous poisoning; this is really a fatty infiltration.

If the liver and kidneys are far advanced in fatty change, the walls of the heart will usually be found yellow, flabby, and brittle.

In the hemorrhagic form of small-pox, besides these changes you will find small hemorrhages in nearly all the

viscera, with ecchymoses of the serous membranes and fluid blood in the cavities. Every mucous membrane may be the seat of a hemorrhage.

The characteristic anatomical lesion of small-pox is to be found upon the mucous membranes and upon the skin. This lesion is usually spoken of as the eruption. It does not differ essentially in the different varieties of the disease; the modifications which are met with are due rather to its duration and order of development than to any difference in the anatomical changes.

If we study closely these surface lesions, we will find that they pass through regular stages of development and decline. The first change that is noticed looking towards the development of this lesion is congestion of the papillæ. In some cases this congestion seems to occur in spots, while in other cases it is quite uniform. The congestion of the papillæ gives rise to the little red spots upon the surface, which are the first to mark the development of the eruption. The papillæ, which are the seat of the congestion, will very soon be found to be surrounded with cells, which are larger than those seen in the normal tissue of the part. These cells very rapidly undergo granular degeneration. Most of these cells have escaped from the blood-vessels or are changed tissue-cells. As these new cells accumulate, they cause the epidermis to become elevated, and as the result of the elevation we have a little papule formed at the point of redness. The papules which are formed at the red point are due to the changes in the surrounding cells, changes in the rete-Malpighii and in the capillaries, and also to a certain extent to new-cell infiltration.

After these changes have taken place, you will notice a serous infiltration upon the surface of the papule, or perhaps into its substance. This serous fluid is simply the serum of the blood which has escaped through the walls of the congested capillaries, and formed upon the top of the papule a little elevation which is recognized as a vesicle.

In a certain proportion of cases, you will find that, soon after the vesicle begins to form, its centre becomes depressed. This depression or *umbilication*, as it is usually

called, has been accounted for in different ways by different observers.

Some explain it by saying that each papule and subsequent vesicle holds imprisoned at its centre either a hair-follicle or the duct of a sweat-gland, and that when this epidermal layer of the papule is elevated by the serous exudation or infiltration, the portion immediately about the hair-follicle or the sweat-duct is held down, and a depression is produced at the exact point where the hair-follicle or duct of the gland may be situated. Another explanation (which I regard the better one) of the umbilication of the vesicle is, that the serous infiltration takes place more rapidly at the periphery of the vesicle than at its centre: consequently, the former becomes more elevated than the latter.

Umbilication of the vesicle is by no means of constant occurrence, as can be readily ascertained by close examination of a number of vesicles.

We have considered those anatomical changes which take place in the papule and vesicle. Now another process commences, pus-cells or white blood-globules from the capillaries migrate into the surrounding tissues and into the vesicles, and as a result the vesicles change in color. In other words, the vesicles become changed into pustules. At the same time an inflammatory process, more or less extensive, is going on in the walls of the pustule, and in the surrounding cellular tissue, which terminates in a destruction of tissue at the point where the papillary congestion first occurred. If only the superficial layer of the skin is involved, the infiltration of pus-cells into the vesicle and the formation of the pustule may take place without extension of the inflammation into the cellular tissue beneath, and necrosis or death of the part will not follow; but, if you have the inflammation extending into the deeper tissues, a slough will be produced, which necessarily will be followed by a cicatrix and pitting.

Remember that pitting is the result of a slough that has been produced by an extension of the inflammatory process into the deeper tissues.

When the cellular tissue becomes involved in inflammation it readily undergoes the sloughing process. This is the reason why we have pus so readily formed and in such quantities, when in any part of the body an apparently slight degree of inflammatory action invades this tissue. I dwell upon this point, for it has a bearing upon the treatment of the pustule, for which a long list of remedies has been proposed with the view of preventing pitting. Prevention of pitting will depend upon the extent of the inflammatory process; if it involves the cellular tissue, pitting will follow in spite of all the applications that may be made. You may prevent pitting if you can find any means of arresting the inflammatory process before it involves the cellular tissue. After the pustule is formed the inflammatory products begin to dry down, and a crust is formed which contracts in the central portion, and the same umbilicated appearance is presented that is seen in the umbilicated vesicle. After a time these crusts are separated by the ordinary changes which occur in the subsidence of an inflammatory process, and recovery is complete, except that there is left behind a cicatrix, which undergoes the same changes as does a cicatricial tissue formed under any other circumstances.

I have now briefly described to you the anatomical lesions of a variola pustule. I would add that these pustules may be formed upon any of the mucous membranes of the body. They are also frequently formed upon the mucous membrane of the stomach, intestines, bronchial tubes, larynx, and upon the conjunctivæ. The surface of the body is the place where they are most abundantly developed. The anatomical changes which take place in the skin and mucous surfaces are similar to those which attend any ordinary inflammatory process. These inflammatory processes are set up by a specific small-pox poison, which carries with it a tendency to produce destruction of tissue at the point where the inflammation is established. In the milder forms of small-pox, pitting does not occur, but in severe forms it is always present to a greater or less degree.

There is nothing specific or essentially different in the

development of the pustules in hemorrhagic small-pox from their development in the ordinary forms of the disease; the only difference is, that their contents are bloody instead of serous or purulent.

In the hemorrhagic variety, larger or smaller hemorrhages take place into the cellular tissues; in the milder forms they take place only in the layer beneath the papillæ; while in the severer forms they take place beneath all the cutaneous layers; even the subcutaneous fat may be infiltrated with blood. No changes in the walls of the vessels have as yet been discovered which will account for these hemorrhages. These extravasations more frequently occur in those cases in which death takes place before the period of pustulation is reached.

ETIOLOGY.—The etiology of small-pox is a subject which at different times has engaged the attention of the profession. At the present day the opinion is almost universal that the disease is propagated only by contagion; that is, that it is a disease which can only be produced by its own specific poison, and is communicable only to persons who are not protected from its influence.

There has been considerable question as to where the virus of small-pox is located. Some claim that it is exclusively in the pustule, and that it is not possible for a person suffering from small-pox to give the disease to an unprotected individual unless some of the virus from the pustule is brought in contact with a cutaneous or mucous surface.

This is a mistake. That small-pox can be conveyed by means of virus taken from a pustule there can be no question; but you may rub the cutaneous surface of an unprotected person with pus taken from a small-pox pustule, and unless there is an abrasion of the surface the poison will not enter the body and the person will not become inoculated with the disease; but if you place the virus in contact with a mucous membrane of an unprotected person he will almost certainly contract the disease. It is equally certain that the disease can be communicated from one person to another by means of the breath and exhalations from the skin. There is no evidence that the disease can be con-

veyed by the discharges from the bowels. Perhaps if a pustule should be developed somewhere along the line of the intestine the discharges may become so contaminated as to have the power of communicating the disease.

Small-pox can also be conveyed from one individual to another through the atmosphere. In the open air the distance of contagion is about two and one-half feet. In a small room the atmosphere may be so contaminated that an unprotected person will contract the disease upon a single entrance into the room.

The disease can be conveyed in clothing, and the poison will remain for a long time in clothing, unless it has been exposed for a considerable time to the air. In other words, there is no doubt but that it is a portable disease. In order that the disease may be transferred by means of the clothing or merchandise, it is necessary that the clothing or merchandise contain the pus or crusts from the small-pox pustules; how long a time may elapse before the virus loses its vitality is not known. There are well-authenticated cases in which it has retained its virulence for more than a year.

No period of life is exempt from the contagion of small-pox; even intra-uterine life is in danger from infection. Rarely does an individual have a second attack. I remember one exception, that occurred in the person of a young Swedish woman, who under my observation passed through three well-developed attacks of the disease; the last attack was the most severe.

Concerning the exact nature of the small-pox virus nothing definite is known.

Some observers claim that the earliest period at which one suffering from this disease can infect the unprotected is the period of suppuration. Others claim that the infecting period is during the stage of desiccation. There are well-authenticated cases, however, which prove to us that infection may take place during any stage of the disease, even during the period of incubation. There is little doubt but that the suppurative stage is the most infectious period.

There are differing views as to the manner in which the

small-pox poison gains entrance into the system ; the most probable of these views is, that it is principally absorbed by the mucous membrane of the respiratory track during respiration, and it is also probable that exceedingly fine particles detach from the pustules and crusts, which are suspended in great numbers in the air surrounding small-pox patients, and that these convey the contagion. There are no facts to sustain the recent views as to the parasitic nature of this contagion.

The length of time which elapses after exposure to, and reception of, the variola contagion before the disease is developed varies from ten to thirteen days. This is called the period of *incubation*, during which the recipient of the poison usually presents no abnormal symptoms. If the poison is introduced into the system through inoculation, only forty-eight hours elapse before the characteristic phenomena of the variola are manifested. It is not known what change takes place in the body of the infected person during this period of incubation. Usually, twelve days after exposure, the person who has contracted small-pox begins to feel chilly ; this feeling of chilliness increases until he has a distinct chill. This has been termed the initial stage, or the stage of initiatory fever.

SYMPTOMS.—The transition from the stage of incubation to that of initiatory fever is sometimes abrupt and sometimes gradual ; usually it occupies two days and is followed by the eruption. In this stage there is greater variation in the intensity than in the duration of the symptoms. The intensity of the symptoms bears no relation to the severity of the attack. Not unfrequently, the most violent symptoms in the initial stage are followed by a mild attack of variola ; while mild symptoms in the initial stage are followed by the gravest form of small-pox. With the chill, which may be more or less severe, there is severe pain in the head and back, especially in the middle of the back ; with this pain there will be a rapid rise in temperature. During the first day the temperature may rise to 104° F., during the second day to 105° F., and by the third day it may reach 106° F. or 107° F. ; in some cases it has been

said to have reached 109° F. With this rise in temperature there will be an acceleration of pulse; it may reach 100 or 120 beats per minute. In the strong and robust person, the pulse will be full and not easily compressed. In females, and in the weak and feeble, the pulse has less volume, but usually is more frequent; it may reach 140 beats per minute.

At the very onset of the disease, the pulse become markedly increased in frequency, and the temperature becomes very much elevated.

At this period, usually, there is more or less nausea and vomiting, and there will be soreness of the throat. This soreness of the throat may have preceded the chill by twenty-four hours, but now in many cases it will be quite severe, and the patient will complain of more or less difficulty in swallowing, and of pain in the pharynx. The extent of the trouble in the throat will depend upon the severity of the attack.

In the severer forms of the disease, by the third or even before the end of the second day, there may be delirium. In all cases, the face will be flushed, the conjunctivæ congested, and there will be throbbing of the carotids. With these symptoms, there will be great restlessness, and an anxious expression of countenance, with somnolence. The respirations will be short, frequent, and labored. Many suffer from extreme vertigo, and in children convulsions are not infrequent. By the evening of the second, or morning of the third day, usually swelling and diffuse redness of the tonsils and soft palate are present; not unfrequently the swelling and redness of the mucous membranes extends into the larynx, causing hoarseness and huskiness of the voice and a stridulous cough.

Some writers describe an initial erythematous rash which precedes the eruptive stage of small-pox. This rash is so rarely met with in this country that it seems to me to be an accidental occurrence rather than a symptom of the initial stage of the disease.

During the fever of invasion patients are languid and weak in proportion to the severity of the fever. Fre-

quently, within twenty-four hours, after the ushering-in chill the strongest and most vigorous will be unable to get out of bed.

There is always loss of appetite; nausea and vomiting are frequently present. If vomiting occurs it is present at the very beginning of the initial fever, and continues with great obstinacy throughout its entire course. In the hemorrhagic variety the matters vomited may contain blood.

Stage of Eruptions.—By the third day of the disease, at least after the initial fever has continued three full days, an eruption will make its appearance upon the face, especially along the edges of the hair.

I will describe the eruption as it develops in a moderately severe case of discrete variola. It first appears in the form of slightly elevated maculæ. These are of a pale red color, varying in size from a millet-seed to a pin's head, or even larger. These little red spots look very much like flea-bites. In most cases the forehead, nose, and upper lips are covered first. If you closely watch them you will find that they gradually increase in size; the increase is attended by a sensation of itching and burning of the surface. Usually, about twelve hours after their appearance upon the face, similar small red points appear upon the body and extremities; first on the body, then on the legs and arms, and lastly on the hands and feet. They are always less abundant on the body and extremities than on the face. On the second day of the eruption these spots assume a darker red color, become elevated and distinctly papular. On the third day they become more conical in shape, and at their apex a vesicle is formed, which gradually enlarges until the fourth or fifth day, when they reach the size of a small pea, and are spherical in shape.

In a majority of instances, as they enlarge, a depression is formed, which gives to them an umbilicated appearance. At the centre of the depression the opening of a hair-follicle or sweat-gland will often be found. The appearance of eruption is attended by a subsidence of the febrile symptoms, the patient no longer complains of pains in the head and back, the temperature falls two or three degrees,

and the pulse diminishes fifteen or twenty beats in frequency.

Stage of Suppuration.—About the sixth day of the eruption the contents of the vesicle, from the admixture of pus-corpuscles, gradually become turbid, and by the eighth day the pustules become fully formed, and the disease enters on the stage of suppuration. The integument in the immediate vicinity of the pustule now becomes red and tumefied, each pustule being surrounded by a broad red base, and where they are thickly set they become confluent. The face swells to a shapeless mass, and the patient becomes frightfully deformed. The itching now becomes almost unbearable, and causes the patient to scratch himself, thus causing ultimate disfigurement. During this period a characteristic sickly odor is emitted.

As I have already stated, the eruption appears on the trunk and extremities a day or two later than on the face, and on these parts it passes through its stages two or three days later than it does on the face; consequently, suppuration may be complete on the face while it is still taking place on the extremities, and the eruption may be perfectly discrete on the trunk, while it is confluent on the face.

About the eighth or ninth day of the eruption the pustule is fully formed; the stage of suppuration is complete. Then commence the retrograde changes. The pustule either ruptures, discharges its contents, dries up and forms a yellowish crust, or it shrivels and dries up without rupturing.

This is called the period of *desiccation*.

Stage of Desiccation.—*Desiccation* commences in those parts in which the eruption first appeared. As the drying down of the pustules takes place, the redness and tenderness of the skin lessens, and the countenance begins to assume a more natural appearance. At first the crust adheres quite firmly to the surface, but between the eleventh and fourteenth day of the eruption it is separated from the surface and falls, leaving a stain of a reddish-brown color, with elevated edges and depressed centre, which remains visible for five or six weeks. These spots gradually become lighter in color, until finally, if there has been destruction of the

cutis, a pit will be formed of greater or less depth, of a white color, giving to the face a "pock-marked" appearance, which will remain during the life of the individual.

I have already stated to you that the febrile symptoms gradually increase in severity until the third day of the disease, when the eruption appears and the fever subsides. Then the vesicles form, the formation of which is attended by only moderate fever. On the eighth day the pustules are fully formed, and the suppurative, or, as it is called, the *secondary fever* comes on. This *secondary fever* often commences with a distinct chill, the pulse becomes frequent, the temperature rapidly rises, perhaps reaches a higher elevation than it did during the initial fever, sometimes rising as high as 108° F. or 109° F. ; it reaches its maximum when suppuration is at its height. As desiccation commences, the temperature begins to fall, and by the time the crusts are fully formed the temperature reaches very nearly a normal standard. If the temperature rises again, its rise is due to some complication, such as erysipelas or some phlegmonous process. With the fall of the crusts, the patient's appetite returns and he is able to sleep ; convalescence is now fully established.

LECTURE XXIV.

SMALL-POX.

Symptoms (continued).—Differential Diagnosis.—Prognosis.

I HAVE already given you the history of the symptoms of an ordinary case of discrete small-pox. This may be regarded as a prototype of all varieties. This morning I shall call your attention to the points of difference between the other varieties of small-pox and that variety whose history we have been considering. The dividing lines between these different varieties are not sharply defined; one variety gradually passes into another variety.

It is unnecessary for me to consider all the forms into which this disease has been divided by medical writers; frequently the basis of the division is merely arbitrary. We will therefore confine our attention to the more common and well-recognized varieties.

Confluent Small-pox, or Variola Confluens.—This is a much more severe form of the disease than *variola discreta*. It develops far more rapidly and is much more fatal in its results.

The fever of invasion is usually much more severe, and of shorter duration, frequently not lasting more than forty-eight hours. The eruption spreads rapidly over the entire body, often appearing simultaneously on the face and the other portions of the body. The red dots which mark the first appearance of the eruption are very numerous, especially on the face and hands; on the first day of their appearance

they are almost confluent. On the second day the skin is intensely red and swollen, and so thickly studded with large flat vesicles that they rapidly unite, suppuration speedily follows, and flattened, yellowish-colored confluent patches are formed upon a dark, reddened, swollen skin. Gradually these patches run together over a still larger surface, and the epidermis is elevated in the form of large, flat bullæ, which are filled with a sero-purulent fluid. In this way the entire skin of the face is covered by an immense bulla, and the patient is as unrecognizable as though he wore a mask. While the eruption may be completely confluent on the face and hands, on other parts of the body it remains distinct, and never becomes confluent except over limited spaces.

The period of desiccation is slowly reached. Large concentric crusts are formed over the confluent patches; these adhere firmly to the skin, while beneath them suppuration of the papillary layer continues. The true skin is more or less extensively destroyed, and when the crusts have fallen, there is left extensive loss of substance in the cutis, giving rise to pits and ugly scars, which have a tendency to contract, often producing permanent and unsightly disfigurements. In this variety of small-pox, the eruption is often confluent upon the mucous membrane of the mouth and throat; it may involve the mucous membrane of the posterior nares, and extend into the larynx. In some cases the attending pharyngitis is so severe as to render deglutition impossible. The pharyngeal inflammation is submucous, and is frequently accompanied by more or less enlargement of the parotid and sublingual glands. When this condition exists there is danger of the sudden development of *œdema glottidis*, for the occurrence of which you should be on the watch. During the year that I had charge of the Small-pox Hospital, there were three cases in the hospital of *œdema glottidis*; one case terminated fatally before I reached the patient; life was saved in the other two cases by the performance of laryngotomy.

In confluent small-pox the severity of the constitutional symptoms corresponds to the severity of the local manifes-

tations. The temperature during the initial fever often reaches 106° F. or 107° F., and in very severe types of the disease it may rise as high as 110° F. The pulse is correspondingly frequent and feeble. After the appearance of the eruption the temperature falls slowly to 103° F. or 104° F., where it remains until the stage of suppuration is reached; then it again rises, in some cases even higher than during the period of invasion. Violent delirium is very frequently present during the fever of invasion, as well as during the period of secondary fever, and not infrequently patients pass, quite suddenly, into a state of coma. Uncontrollable vomiting and obstinate diarrhœa are not infrequent, coming on during the fever of invasion and continuing throughout the course of the disease. In all severe cases typhoid symptoms manifest themselves soon after the appearance of the eruption, and patients often lie for days in a semi-conscious state, with dry, brown tongue, subsultus, a low muttering delirium, and all the attendant phenomena of intense nervous depression. In all severe cases albumen appears temporarily in the urine.

Complications occur much more frequently in confluent than in discrete small-pox. Inflammations of the serous membranes, especially pleurisy and pericarditis, are the most common. Croupous and catarrhal pneumonia frequently complicate the severe bronchial inflammation from which so few patients with confluent small-pox escape.

VARIOLA HEMORRHAGICA.—There is another form of small-pox which can hardly be regarded as a distinct variety, but rather as a modification of those varieties which have just engaged our attention, and which has been called *hemorrhagic variola*. It differs from the varieties already described, not in the manner of its development as far as the initial fever is concerned, but in the appearance of the eruption. This hemorrhagic tendency is often manifested as early as the first appearance of the eruption, by the dark color which the eruption assumes. Sometimes the papules become hemorrhagic from the very moment of their development; at other times they first become vesicles, and then become hemorrhagic. Again, at other times, the hemor-

rhage first shows itself after the vesicles become pustules. In some cases the eruption over the whole body becomes hemorrhagic; in other cases it is hemorrhagic in spots. In the majority of the cases of this variety, however, the eruption becomes hemorrhagic as soon as the papules have attained the size of a lentil, and the hemorrhagic change comes on slowly, generally commencing upon the lower extremities. Petechiæ and ecchymoses usually appear between the points of eruption, if the small-pox is of the discrete variety.

In connection with the hemorrhagic eruption, at the same time you will have hemorrhages from the various mucous membranes of the body—from the mucous membrane of the nose, perhaps from the bronchial mucous membrane, and sometimes large ecchymotic spots may be seen upon the mucous surfaces of the mouth and throat.

It is rare for this form of small-pox to reach the stage of suppuration, for before this stage is reached patients with hemorrhagic small-pox sink and die, either from the overwhelming influence of the small-pox poison, or from the exhaustion caused by extensive hemorrhage.

In females profuse menorrhagias are of frequent occurrence, and often are so extensive as to endanger life.

During the initial stage of this variety of small-pox, the constitutional symptoms do not differ from those which attend the development of the other forms of this disease. It is impossible, from their character and intensity, to predict, with any degree of certainty, the subsequent development of hemorrhagic variola. It has been said that the pains in the back and limbs are more severe; but these are not sufficient to characterize this type of the disease. Frequently the fever of invasion is exceedingly violent, while during the eruptive period, and during the entire subsequent course of the disease, the temperature is comparatively low; sometimes during the entire course it does not rise above 102° F. In striking contrast with the low temperature is the frequency of the pulse. In those cases in which extensive hemorrhages have occurred, the temperature often falls below the normal standard, while the pulse

ranges from 140 to 160, and is exceedingly feeble in character. Only when a comparatively few of the vesicles become hemorrhagic does the case terminate in recovery.

Before describing the modifications of small-pox produced by inoculation and vaccination, I will complete the history of those varieties which have already engaged our attention.

DIFFERENTIAL DIAGNOSIS.—The first question that comes to us under this head is, How early can small-pox be recognized? One who has seen very many cases of the disease may be able to reach a diagnosis on the third day, that is the first day of the eruption, although at that time there is nothing characteristic about the eruption or the ushering-in symptoms. It is, however, better and safer to wait until the second or third day of the eruption before committing yourselves to a positive diagnosis, for there is little to be feared from infection until the vesicles are fully formed; then you may be positive in regard to your diagnosis.

The exanthematous fever which, in its early stages, and on account of its eruption, is most liable to be taken for small-pox, is measles.

Usually the eruption of measles is so distinct and well defined that you will not mistake its true character; but there are cases of measles in which the eruption presents an appearance closely resembling the first appearance of the eruption of small-pox. Such cases are not altogether infrequent. A number of cases of measles came under my observation while in the Small-pox Hospital that had been sent by physicians to the hospital as cases of small-pox.

If you defer making a diagnosis until the vesicles are fully developed, you need make no mistake of this kind.

In measles there is coryza, a cough, sneezing, redness and suffusion of the eyes. These symptoms are not present in small-pox.

In small-pox, when the stage of eruption is reached, the temperature falls; while in measles, when the eruption appears, the temperature continues to rise. The range of temperature is higher in small-pox than in measles. In these respects the two diseases differ sufficiently to enable

you to make a differential diagnosis. Again, if you wait until the vesicles become umbilicated, it will hardly be possible that you should make a mistake in diagnosis.

During the period of initial fever it is possible to mistake small-pox for typhus fever. In both diseases there may be delirium, pain in the head, vertigo, high temperature, and evidence of great disturbance of the nervous system. I know of no symptom which will enable you to make a positive diagnosis during the very early period of the disease. Of course, if typhus fever is prevailing, or if small-pox is prevailing, and the patient has been exposed to either one of these contagions, you will be able to make a diagnosis without much difficulty. Usually there is greater loss of muscular power in typhus fever than in small-pox, but this symptom is not always well marked. By the third day, the appearance of the eruption upon the face, where it is first seen, settles the question of diagnosis. The eruption of typhus fever is first seen upon the abdomen, and it may extend over the whole body without appearing on the face. It rarely appears before the fifth day of the fever. If, therefore, you wait until the eruption appears, the differential diagnosis between small-pox and typhus fever can be readily made.

Meningitis is another disease which small-pox, in its initial stage, resembles. I have seen a case of small-pox treated for several days as a case of meningitis. There is always considerable cerebral disturbance, and a full, hard, bounding pulse in the initial stage of small-pox. Photophobia and intense pain in the head, as also nausea and vomiting, may be present in both diseases. Unless it may be the expression of the face, there is often no distinguishing mark between the two diseases in their early stages. In meningitis there is usually a pale, anxious expression of countenance, whereas early in small-pox the face is flushed, and day by day the flush deepens until the eruption appears. There is often a uniform redness over the entire surface of the body in confluent small-pox when the eruption appears, or at least that portion of it where the eruption makes its appearance.

On the appearance of the eruption the differential diagnosis between these two diseases is readily made. I wish to impress you with the fact that it is much better to wait in all doubtful cases, perhaps in every case of small-pox, until the eruption appears before attempting to make a diagnosis.

It is an unfortunate occurrence whenever a patient, who is not sick with small-pox, is sent to a small-pox hospital, and it is equally unfortunate whenever a small-pox patient is retained in a family or neighborhood a sufficient length of time to expose the remaining members of his own family or other families in the neighborhood to the contagion of this disease; but there is little danger of infection until the vesicles are fully formed.

PROGNOSIS.—The prognosis in any case of small-pox depends upon the amount of the eruption; the more abundant the eruption, the greater the danger to life. The prognosis also depends upon the type of the disease. Unless some complication arises, the majority of cases of discrete small-pox recover; while of confluent small-pox, which is a much graver disease, nearly one-half the cases prove fatal.

The best record obtained in the Small-pox Hospital on the island was one death in every five cases. In the hemorrhagic variety, whether discrete or confluent, a fatal termination is almost inevitable. Only a very few cases of the hemorrhagic variety recover, and when recovery does take place it is only reached after the patient has passed through an apparently fatal condition of coma.

The ratio of mortality is always lower at the end than at the beginning of an epidemic. It is more fatal in the summer than in the winter.

The age of the patient greatly influences the prognosis. In infancy and in extreme old age the ratio of mortality reaches its maximum. Among adults, the prognosis is worse in females than in males. In the intemperate the prognosis is always bad, for with this class of persons the disease is liable to assume a hemorrhagic type. The intemperate die in discrete small-pox when the temperate will with almost certainty recover. In the overworked and

badly-nourished the prognosis is bad. Robust and healthy persons pass through a severe type of the disease much more safely than those enfeebled by syphilis and other chronic forms of the disease.

The severity of the fever of invasion is not a safe guide in prognosis. Sometimes a severe initial stage precedes a mild form of the disease; sometimes patients with this disease pass into a state of complete unconsciousness, remain in that condition for some time, then the eruption begins to change in color, and finally recovery takes place. Such cases, however, are exceptional.

However well developed the eruption may be, or however well filled the vesicles, you must remember that the eighth day is the commencement of the suppurative fever, which is the period of the greatest danger. Upon this day you may find your patient passing into a state of collapse, the result of the depressing influence upon the nervous system produced by the large extent of surface involved in the suppurative process. If patients do not die until the second week of the disease in most cases the fatal result is due to exhaustion, although death may occur from complications. Usually they pass into a typhoid condition, the result of the excessive drain upon the system while the suppurative process is going on. Secondary syphilis is occasionally developed during the period of desiccation. All such cases that have come under my observation have proved fatal. The most frequent complications which cause death are those which occur in the throat and air-passages. In some instances swelling of the glands of the neck and mucous membrane of the throat takes place to such an extent as to seriously interfere with deglutition and respiration. When this occurs it becomes a great element of danger, and materially affects your prognosis. The tongue may become swollen to such an extent that the patient will be unable to protrude it, or, being able to protrude it, will not be able to retract it. Under such circumstances deglutition is almost impossible, and, as I have already stated, oedema glottidis is liable to occur. You may have laryngeal ulcers, and ulcers occurring in the trachea and in the bronchial tubes. These

may give rise to changes which will so interfere with respiration as to cause the death of the patient. Death may also occur from general bronchitis or pneumonia. Perhaps the most dangerous complication is acute fatty degeneration of the kidney. Whenever, in the course of the disease, the urine becomes scanty and high-colored, but especially when it becomes so at the commencement of the secondary fever, you may then be certain that you have kidney complication. Under these circumstances your patient may pass into a condition in which convulsions will be developed, and coma and death ensue.

Before leaving this subject I will call your attention to a case of confluent small-pox which came under my observation about one month ago. I make mention of this case that I may impress upon you the importance of one symptom as regards prognosis, that is, the *abundance* of the eruption.

I was called in to visit a gentleman who was in the initial stage of the disease. I had charge of him up to the third day of his illness. At that time an abundant small-pox eruption had made its appearance. He then passed into the hands of a younger physician, who seemed amazed when I said to him that I thought the patient would die. A few days later the physician informed me that the patient was doing well, and he thought I had made haste in my prognosis. In reply I said, "wait until the *ninth* day." Upon the *eighth* day I saw my professional brother again. He then remarked that the patient was very much worse, and he was afraid he was going to die. He died a short time after our last conversation. Now, the only symptom which led me so early to make an unfavorable prognosis was the *abundance* of the eruption.

In the hemorrhagic variety of small-pox usually the stage of suppuration is not reached—the patient dies before that period on account of the extensive changes which take place in the blood. Under such circumstances you are liable to have complete suppression of the urine, or, at least, sufficiently complete to give rise to uræmia in addition to the small-pox poisoning.

TREATMENT.—We now come to the treatment of small-pox. The first question that arises under this head is, have we any means by which we can arrest its development after the initial fever has been established? In vaccination, properly performed, we undoubtedly possess a means by which we may prevent one from contraction of the disease when exposed to its infection.

But the question now arises, have we any power to arrest the development or mitigate the severity of the disease after the initial fever is established? No reliable affirmative answer has been given to this question. It has been proposed to accomplish this by blood-letting, emetics, diaphoretics, purgatives, cold-baths, and more recently by the subcutaneous injection of the vaccine-virus. All of these means have been tested and have failed to accomplish the desired result.

The assertion that large doses of quinine, given during the stage of invasion, will shorten the duration and modify the course of the disease is verified only by the experience of its author.

Quite recently, it has been claimed that carbolic and salicylic acid will destroy the septic poison of variola, and thus shorten and modify its course. My own experience as regards their use has not been sufficient to decide the question for myself, but I am unable to find any statistics which sustain such an assertion.

During the fever of invasion all that you can do is to treat special symptoms.

Place the patient in bed in a large, well-ventilated apartment; if possible keep the temperature of the room below 60° F. I remember that, in the Small-pox Hospital, those patients did best who were placed in barracks which were so open, that frequently, during the winter months, when I made my morning visit, I would find little snow-drifts on the floor between the beds.

When the body temperature ranges as high as 107° F., or 108° F., it may be necessary to employ cold to the surface, and to give antipyretic doses of quinine to reduce the temperature. If the headache is severe and the face flushed, iced compresses and ice-bags to the head will usually afford

relief. If the vomiting is severe and constant, iced carbonic acid water may be given, and if the vomiting is attended by great restlessness, hypodermic injections of morphine are indicated. Administer such food as can be readily assimilated. I have found nothing better than iced milk and seltzer water. If the bowels are constipated it is well to relieve them by enemata of cold water.

In those cases in which the eruption is tardy in making its appearance, and the temperature is higher, sometimes, if the patient is kept in a warm bath for fifteen or twenty minutes, the development of the eruption is hastened.

When the eruption has appeared, the measures to be employed will vary with the character of the eruption. The milder forms of discrete variola require no interference. In the severer forms the attendant symptoms will decide the means to be employed.

Sooner or later, sometimes very early in the severer forms of the disease, you will find your patient sinking from the depressing effects either of the small-pox poison or of the suppurative process which is taking place upon the surface of the body. Under such circumstances you will be compelled to resort to the use of stimulants. There is no question but that the free use of stimulants for a few days, just at the period of suppuration, in very many cases does much to save life. At this time you may find your patient with a dry tongue, a frequent, feeble pulse, blue lips and finger ends, giving evidence that he is rapidly passing into a state resembling that met with in the latter stages of typhoid fever. Active delirium is frequently present; the patient insists upon getting out of bed. Under these circumstances, the life of your patient will often be saved by the judicious use of stimulants. If the delirium is excessive, hypodermics of morphine may be combined with the administration of stimulants. During the stage of desiccation, warm baths employed every day, or every other day, give great comfort, and assist in the falling of the crust. After the baths, the surface should be freely oiled.

Complications will be treated according to the general rules which govern their treatment. If abscesses occur

in the subcutaneous tissue, they should early be freely opened.

We are powerless when we come to deal with the hemorrhagic form of small-pox. Although tonics and stimulants have been highly recommended, they do little good. Transfusion has been proposed and practised with no definite results. If the mouth and pharynx are very much involved, and there is difficulty in deglutition, ice-cold carbonated water with a weak solution of mur. tinct. ferri used as a gargle will often give great relief. Sometimes the stronger antiseptic gargles, such as carbolic acid and permanganate of potash, will be of service.

There is still one point in the treatment of small-pox which is deserving of attention, and that is, what means may be employed to prevent the pitting, especially upon the face, which is so frequent an accompaniment or the result of small-pox? As I have already stated, the eruption first makes its appearance upon the face; there it is usually most abundant, and is most liable to be followed by pitting, and there it passes more quickly through all its stages than upon any other part of the body. In order to prevent the pitting, it has been proposed by some to exclude light and air from the surface covered by the eruption. For this purpose a great many substances have been employed, such as collodion, gutta-percha, certain forms of plaster, liquid paper, etc., etc. All these substances are to be so applied as to form a mask for the face, which should completely exclude light and air from the surface.

You will recollect that I stated that the pitting was due to the formation of a slough, and that the slough was seated in the areolar tissue, and that if by any means you can so interfere with the inflammatory process as to prevent the formation of a slough, you will prevent the pitting. It was claimed by those who advanced the theory, that excluding light and air prevented pitting; that it did this by preventing the occurrence of sloughing.

At the time when I had charge of so many small-pox patients, I took pains to test all those applications, which at that time had been and are still recommended for that pur-

pose, and I satisfied myself that about the same results were obtained in the use of every remedy, and in no case was pitting prevented. Certain patients were much more scarred than others, but that was the natural result of the disease.

Some have proposed to coagulate the serum in each vesicle by means of iodine or nitrate of silver, and so arrest the inflammatory process and prevent pitting. But the use of these means has been attended by the same unsatisfactory results. The only means which I found of certain value was simple cold-water dressing applied over the face, after having ruptured each vesicle before it became a pustule. In this way, I was able to diminish the intensity and extent of the inflammation. This plan of treatment I adopted in twenty cases of confluent small-pox, and it not only gave the patients very great comfort, relieving them to a certain extent from the intense itching, thus avoiding rupture of the vesicles by scratching, but in not a single case that recovered was there bad pitting. In the treatment of small-pox, the prevention of pitting is of greatest importance to a certain class of patients, especially young unmarried females.

LECTURE XXV.

SMALL-POX.

Treatment (continued). — Inoculation. — Vaccination. — Varioloid.

WE will now consider the two recognized methods for rendering small-pox poison so innoxious that, when one has been exposed to its influence, he will be perfectly safe from infection. These two methods are known as *inoculation* and *vaccination*.

Inoculation was first introduced into England in the year 1781, by Lady Montague, who first practised it upon her own child, she having become familiar with the practice while travelling in Italy, where the practice undoubtedly originated. Subsequently it was quite generally practised throughout Great Britain. Pus from a small-pox pustule was introduced beneath the epidermis of one who had been prepared, by diet and general hygienic measures, for the safe development of the disease. It was claimed that the disease resulting from inoculation was a modified small-pox, differing from the original disease in that it ran its course more rapidly, was attended by the formation of a fewer number of pustules—perhaps no more than twenty or thirty upon the entire body—and was said to rarely terminate fatally, the ratio of mortality being about one in one hundred. The patient who had the disease in this manner was equally protected with those who had the disease in the ordinary manner, being exempt from a second attack.

The disease developed by inoculation passed through the regular stages of a case of ordinary small-pox—that is, there was the initial fever, the eruption, and the desiccation, one stage following another in regular succession. This procedure was found more or less unsatisfactory, for it had its disadvantages; there was danger in it, and inoculated persons could communicate small-pox to others.

During the latter part of the last century, Sir William Jenner observed that, in some of the northern counties of England, persons employed in dairies, who suffered from a certain form of ulcer upon the hands, did not contract small-pox when exposed to the influence of its poison. He also found that these ulcers found upon the hands resembled pustules found upon the udder of the cow, and seemed to have been caused by contact with them. Jenner made a thorough investigation of the subject, and arrived at conclusions sufficiently satisfactory to himself to warrant the experiment of taking matter from one of these pustules found upon the udder of the cow and introducing it into the arm of an individual who was supposed to be unprotected from the contagion of small-pox. After the sore upon the arm had run its course, he exposed the individual to the infection of small-pox, and in this way he established its protecting power.

His first experiment was made in the year 1791; but it was not until six years afterwards that the experiment was repeated by any other person. During these six years it is probable that no member of the profession ever received more anathemas or more scurrilous abuse than did Jenner. He was attacked by the leading physicians and surgeons of Great Britain, and persecution and ridicule so followed him, that placards with caricatures of Jenner were posted throughout the streets of London and the principal towns of Great Britain.

Jenner kept steadily at work and repeated his experiments, until he became fully convinced that by vaccination perfect protection could be obtained against small-pox. Within the short space of six years Jenner compelled the profession to admit his statements and adopt his practice,

and, within the five or six years following its first recognition, the practice of vaccination became uniformly recognized and practised.

Vaccination was introduced into this country in the year 1802, by two Boston physicians, and it very soon became the practice of the entire profession. At the present time there is no question among the intelligent portion of the profession, but that vaccination, properly performed, is a perfect protection against the infection of small-pox; if persons contract small-pox after they have been vaccinated you may infer it has not been properly performed. We have no other means of protection.

We will now study the subject of *vaccination*. There are two methods of performing it. One method is to take the *virus* directly from the cow, this is called *bovine virus*; the other method is to take the *virus* from a vesicle developed upon the human body, perhaps a vesicle removed from the original by several vaccinations, this is called *humanized virus*. It has been a common practice to use virus taken from a vesicle that was removed from the original vesicle by two, five, ten, twenty or even forty vaccinations, on the supposition that just as perfect protection was afforded as though the vaccine was taken directly from the cow.

Within a few years it has been found that such a large proportion of the population were not protected from the infection of small-pox, and that cases of small-pox were so markedly increasing in number, that a return has been made to the *bovine virus*. To-day, this form of virus is used by a majority of the profession. I use it because when I obtain a perfect vesicle, after its introduction into the system I am convinced that the person is thoroughly protected against the infection of small-pox poison. I never have this assurance when I use the humanized virus.

Dr. Jenner found that there were several pustules developed on the udder of the cow, which closely resembled each other, but that only one contained the virus which afforded protection from small-pox. In obtaining bovine virus it is of the greatest importance that the genuine vesicle be

selected. In order to make the selection it is necessary one should be familiar with the peculiarities of each variety.

Dr. F. B. Foster and Dr. E. H. Pardee, of this city, have given this subject much study, and their experience and facilities enable them to furnish bovine virus which is perfectly reliable.

If humanized virus is used, not only is the protection less certain and less permanent, but there is danger of introducing into the system the infection of other diseases. I have in my possession facts which prove beyond the possibility of a doubt that syphilis can be conveyed from one person to another by vaccination. In two instances, which came under my own observation, it was so conveyed when the humanized vaccine lymph was employed.

Cutaneous eruptions may also be conveyed by humanized vaccine virus, which may cause the development of very extensive and serious cutaneous diseases.

Again, it must be remembered that if any chronic or acute skin disease exists at the time the vaccine vesicle is running its course, the protective power of the vaccination will be altogether destroyed or very greatly modified.

In obtaining vaccine virus for use, both the bovine and the humanized virus should be taken from the vesicle on the eighth day. The lymph should be taken from the vesicle before the inflammatory process has commenced which is to change it into a pustule. A few years ago it was the common practice in this city to use the vaccine crusts, but this practice has fallen almost entirely into disuse because of the great danger of transmitting disease from one individual to another.

Always use the bovine virus when it is possible to obtain it. If compelled to use the humanized virus, use the lymph. You must puncture the vesicle in such a manner that the lymph cannot be contaminated by the blood; this is best done by introducing your instrument parallel with the arm. The lymph which flows from such a puncture can be preserved upon the convex surface of a piece of quill, and conveyed from one individual to another. Vaccine virus secured from the human arm in this manner is less liable

than any other form of humanized virus to do permanent harm to the vaccinated individual.

The vaccine virus is usually introduced by scarifying the surface, so as to redden it, scarcely drawing blood; then the surface of the quill containing the virus is applied to the scarified part, or the lymph is conveyed from one to the other by direct transmission. The operation is simple, and one with which you are doubtless familiar. It is not necessary for me to say anything in regard to the manner of performing it.

Any irregularity in the development of the vesicle destroys in a greater or less degree its protecting power.

When an individual has been previously vaccinated, it is liable to run an irregular course. A primary vaccination, such as the first vaccination of a child, *should pass through the following regular stages; if it does not, it fails in its protecting power*: Upon the third day after the introduction of the virus you will notice at the point where it was introduced a little red spot. By the fourth day this little red spot will be occupied by a vesicle, and at the commencement of the fifth day you will begin to see around the vesicle a little yellow margin. Now you have a vesicle with a yellowish-white margin at its base. This vesicle goes on increasing in size up to the eighth day, when you will notice that it has become umbilicated, and that there is around it a distinct areola. Previously there has been a trifling areola present; now it becomes very distinct. The vesicle is free from inflammation, and now is the time to take the lymph for the purpose of vaccination, for the vesicle is complete. The lymph should be taken only a short time before using it. Now a change is to take place in the vesicle, and by the next day you will notice that the areola has extended, perhaps so as to measure an inch in diameter; this areola goes on extending itself through the ninth, tenth, and eleventh days, when it will have reached its maximum extent, which may be one or two inches from the vesicle in all directions. It is now of a deep red color. The part over which the areola has spread is more or less elevated, the arm is considerably swollen and painful, and

the adjacent glands more or less enlarged, and tender to the touch. The extent of the enlargement of the gland adjacent to the vaccine vesicle, the axillary gland, if the vesicle is upon the arm, the inguinal, if it is upon the thigh, varies considerably in different persons. In some it is very great, in others it is scarcely noticeable.

The maximum degree of inflammation in the vesicle has now been attained, and there is a distinct infiltration of the tissue about it. On the twelfth or thirteenth day, the red areola begins to decline, the swelling diminishes, and the vesicle, or, more properly speaking, the pustule, ruptures, and the contents escape. The rupture belongs to the natural course of the vaccine vesicle, and is independent of mechanical violence. From this time the inflamed areola becomes less and less distinct, and by the fourteenth or fifteenth day the crust has assumed a dark, brownish appearance, which goes on deepening, until you find on the seventeenth day a deep-brown crust having a central depression and no areola of inflammation. It may be attached to the surface only in one or two places, and can be readily removed; if permitted to remain, usually it falls off on the eighteenth day. This is the course pursued by a perfect vaccine vesicle. The shape and size of the crust will correspond to the shape and size of the vesicle, and in this way you will be able to determine whether the vesicle has or has not pursued a regular course. Of course, the crust will vary in shape according to the vaccination; if you make an irregular scarification, you may expect an irregular crust, for you will have an irregular vesicle. If the virus is introduced at a single point, the vesicle will be circular, and the crust that is formed will also be circular, and occupy the exact space occupied by the vesicle; if otherwise, it is evident that the regular course of the vesicle has been disturbed.

So, also, if upon the eighth day you find a pustule instead of a vesicle, you may be certain that the regular course of development of the vesicle has been disturbed, and complete protection is not afforded against the infection of small-pox.

The inflammatory process around the vesicle is usually

more active when the *bovine virus* is used, than when the *humanized virus* is introduced, and there is more constitutional disturbance. Ordinarily, during the development of the vaccine vesicle and pustule, there is but little constitutional disturbance; this is usually self-limiting, and not sufficiently severe to require treatment. About the eighth or ninth day, the person vaccinated may feel a little chilly, and have severe headache; in most cases there is a slight rise in temperature.

The regular course of the vaccine vesicle may be interfered with by the occurrence of an erysipelatous inflammation, and if such an inflammation does occur during the course of its development, it entirely destroys the protecting power of the vaccination.

Again, if a large quantity of pus has been discharged, and healing of the ulcer does not take place for two or three months, it is evident that something besides *genuine* vaccine virus has been introduced into the arm. In other words, such a vaccination has not pursued a regular course and is not protective. As I have already stated, the presence of a vesicular eruption upon the surface at the time vaccination is performed will interfere with its development, therefore I would advise you never to vaccinate one who has an eczematous eruption upon any part of the body, unless he has been exposed to the contagion of small-pox, for it is very probable that the vaccination will not be a protective one.

It is better never to vaccinate a person having any form of skin disease, especially if the eruption is vesicular in character. The best time for the performance of vaccination is in infancy, between the third and fifth months.

Revaccination should be performed after puberty, and always after or preceding a new exposure to the contagion of small-pox, for the period during which revaccination will afford complete protection is not the same in each individual. In some cases a single vaccination will afford complete protection for a lifetime. In other cases it is necessary to frequently repeat the vaccination, perhaps every two years, in order to secure the desired protection.

Not unfrequently persons are astonished when the re-vaccination runs a regular course, for they suppose themselves perfectly protected against the contagion of small-pox.

The question has been raised, If vaccination be performed previous to an attack from any severe disease, will not the protecting power of the vaccination be destroyed by that disease? Certain facts seem to indicate that such is the case.

Again, is it necessary to repeat vaccination in order to secure its protecting power? To explain my meaning. There is no question but that a child may be repeatedly vaccinated, and after each vaccination may have some sort of a local manifestation, but he will never have but one perfect vaccine vesicle. If the primary vaccination runs a regular course, it affords protection, and the second introduction of the virus seems to me to be unnecessary, as it simply develops an irregular vaccine vesicle. Nor does the introduction of the virus at two places at the same time seem to be necessary, for one perfect vesicle is sufficient.

The next question which presents itself is, What kind of disease is that which is developed in individuals who are protected by vaccination, when they are exposed to the infection of small-pox?

Unquestionably it is a modified form of small-pox. It has received the name of *varioid*.

VARIOLOID.—This is a disease—the result of an exposure to the contagion of small-pox—which would be small-pox, had the person exposed to this contagion never been vaccinated. During every epidemic of small-pox you will meet with a certain number of cases, concerning which you will be in doubt whether to call them cases of *variola* or *varioid*. Certain persons who have never been vaccinated, on account of their naturally slight susceptibility to the infection of small-pox, may have such a mild form of *variola* that it is difficult to distinguish it from *varioid*.

There are certain points of resemblance between *varioid* and *variola*, and there are certain marked differences. *Varioid* differs from small-pox in the rapid development and decline of the eruption, in the small number of the pustules,

and in the short time required for the formation and separation of the crusts. The entire period of the eruptive stage often does not last more than a week. Rarely are there cicatrices or pits after the disappearance of the eruption.

In varioloid and variola the pustules pass through similar stages. We first have the small red spot, then vesicles form, often within twelve hours after the appearance of the eruption. These vesicles rapidly increase in size; sometimes they are umbilicated; by the end of the third day their contents sometimes becomes purulent, without any tumefaction of the surrounding skin. On the fifth day desiccation commences, and is often completed by the seventh day. The majority of the pustules simply dry up, without previously bursting, forming brown crusts which are thinner and smaller than those of variola.

In varioloid you rarely have the regular period of development such as you have in variola. In variola there is the period of eruption, during which the vesicle is perfected; this is succeeded by the period of suppuration, then by desiccation, about fourteen days being required to complete the process; while in varioloid the course of the eruption is irregular, and is usually completed within one week.

Again, in varioloid there is but little constitutional disturbance after the appearance of the eruption. It resembles variola in the severity of the symptoms during the period of invasion, during which time you will not be able to determine whether the case is one of varioloid or one of small-pox. If you are watching, lest small-pox may be developed, then you may be led to suspect its advent from the severe pain in the head and back, and from the general febrile disturbance following an exposure to the infection of small-pox; but as soon as the eruption appears there is an entire cessation of all the active febrile symptoms. During the period of invasion varioloid may be said to very closely resemble variola.

When an individual is exposed to varioloid, the most severe case of confluent small-pox may be the result. This fact proves that varioloid is a modified form of small-pox

which has been produced by vaccination. It is now generally conceded that varioloid is small-pox having a shorter duration and a milder course than usual.

You may say we modify small-pox by inoculation. We do not. There is the same regular development of the disease after inoculation that we have in the ordinary form of small-pox; we only modify its intensity; while by vaccination we not only lessen the severity of the disease, but we are able to so modify the stages of its development as to shorten its duration.

PROGNOSIS.—Usually the prognosis is good. The diagnosis is readily made. The rapidity with which the vesicles are developed, their shorter duration, the subsidence of the fever, and the appearance of the eruption, together with the usual duration of the attack, are sufficient to distinguish it from variola.

TREATMENT.—The treatment for varioloid is the same as for a mild or modified form of small-pox. The patient should be placed in a large, well-ventilated room, quarantined the same as though suffering from variola. If the form of invasion is severe, saline cathartics may be administered. When delirium is present, and the pain in the back is very severe, the moderate use of opium is admissible.

As soon as the eruptive period of varioloid is reached no treatment is required; the patient passes on to a rapid and complete convalescence.

Before leaving the subject of variola, I will refer to a few complications which do not belong to its natural history.

As I have already stated, there really is no dividing line between the local affections of this disease and most of its complications. Bronchitis, more or less severe, accompanies nearly all cases. In some it leads to catarrhal pneumonia, the occurrence of which is always attended with danger. Pleurisy and pericarditis occasionally occur as serious complications.

Laryngeal inflammations are a part of its history. When the laryngitis is accompanied by extensive ulceration of the laryngeal mucous membrane, or when acute œdema of the

glottis is developed, or when it assumes a diphtheritic character, you have developed a series of complications which often quickly destroy life.

Meningitis and cerebral complications are not of common occurrence in variola, although acute meningitis and œdema of the brain do sometimes occur ; so that when very active delirium or sudden coma come on during the eruptive stage of the disease there is reason to fear their development.

A severe form of conjunctivitis may occur, which is sometimes attended by the development of pustules on the palpebral conjunctiva or upon the cornea. When they develop on the cornea, perforation, iritis, and suppuration of the globe may cause destruction of the eye.

In hemorrhagic small-pox hemorrhages into the retina sometimes occur, causing sudden blindness.

Suppurative otitis may occur and may be the cause of partial or complete deafness.

Pyæmia is a very rare complication of variola, although during convalescence superficial cellulitis, terminating in abscess, is not infrequent.

In severe cases, during convalescence, œdema of the feet, due to anæmia, is frequently met with, but I have never regarded it as of serious import.

LECTURE XXVI.

SCARLET FEVER.

Introduction.—Morbid Anatomy.—Etiology.—Symptoms.

THIS morning we come to the study of the second in the list of exanthematous fevers, namely, *scarlatina* or *scarlet fever*. This name has been given on account of the bright red appearance of its eruption.

Scarlet fever is an inflammation of the tegumentary investment of the entire body, both cutaneous and mucous, accompanied by a fever of an infectious or contagious character.

It is a disease of childhood, but may occur at any age.

Its development and course is divided into periods: *First*, the *period of invasion*, which lasts from twenty-four to forty-eight hours; *then*, the *period of eruption*, lasting from five to seven days; afterwards, the *period of desquamation*, during which the entire epithelial surface is removed.

Some authors have classified this disease according to its severity; others according to the prominent organs of the body which are involved; others according to the prominent phenomena which attend its development.

The more common classification, and certainly the simplest, is that which divides it into *scarlatina simplex*, *scarlatina anginosa*, and *scarlatina maligna*. I shall adopt this last classification.

Scarlet fever has many different types; these are as unlike as some of the distinct types of fever.

MORBID ANATOMY.—There are no characteristic anatomical lesions of this disease, except those changes which have their seat in the skin and mucous membranes. I stated that the characteristic anatomical changes of variola were to be found in the eruption which followed regular stages of development, so in scarlet fever the eruption is the distinguishing lesion.

The eruption makes its appearance on the second or third day after the commencement of the febrile symptoms.

At that time it consists of very numerous and closely aggregated points about the size of a pin's head; between these the skin is of its natural color. In typical cases, these points are equally distributed over the entire body except the face. These red spots are usually circular in shape, slightly elevated above the surrounding skin, and so close to each other that they give a confluent redness to the entire surface. In mild cases the red points remain isolated, and do not become confluent; as the eruption develops these red points unite. In severe cases the skin becomes turgid and swollen, and presents a uniformly red and glistening appearance. In malignant cases the hyperæmia of the skin is often accompanied by more or less extensive hemorrhages, causing petechiæ and extensive ecchymosis.

The eruption gradually increases in redness to a certain point, which is not the same in all cases, then remains unchanged for twelve or twenty-four hours, after which time the redness slowly passes away. During the course of the disease the color often changes with the exacerbations and remissions of the fever. As a rule, the degree of redness depends upon the intensity of the fever, and may vary from a pale red to a dark scarlet color. If the respiration becomes impeded, the eruption assumes a bluish-red hue. During the first forty-eight hours after the appearance of the eruption, when the respiration is unimpeded, the redness completely disappears under firm pressure, and reappears as soon as the pressure is removed. After this period, the pressed point does not entirely lose its red color.

In a certain proportion of cases, the eruption only appears in spots on the surface of the body, on the trunk, or face, or about the flexors of the joints. When it only appears on the face the diagnosis is difficult. In addition to the cutaneous hyperæmia which gives the redness to the surface, there is more or less serous exudation into the "rete Malpighii," which is followed on the decline of the redness of the surface by an abundant epidermic exfoliation. This exfoliation marks the period of desquamation, which may immediately follow the decline of the redness or it may be delayed a few days. This is due to an excessive production of newly-formed epidermis, and the process may last only a few days, or if the eruption is abundant it may continue for several weeks, and may recur a second time on the same surface. After the desquamation has ceased, it does not reappear, except in cases of relapse; these are followed by renewed and sometimes by a very complete desquamation.

In connection with these cutaneous changes the scarlatina poison causes changes in the mucous membrane of the mouth and throat, the most frequent of which is catarrhal pharyngitis, which at first gives to the mucous surfaces of the tonsils and pharynx a red, swollen, and dry appearance. After a little time, these mucous surfaces become covered with a tenacious mucus. Upon the reddened mucous membrane, small elevations arise, like the smaller follicles in an ordinary catarrh. In mild cases, all these changes disappear in a few days; in the severer cases, the mucous surface assumes a dark, livid color, the parts become more or less œdematous, and are covered by an abundant secretion. The œdema may be so extensive as to render deglutition difficult; often the tonsils are so swollen that they touch each other. Besides the redness and œdema of the mucous membrane of the mouth and throat, there is often inflammation of the parotid and sublingual glands as well as of the connective tissue of the neck. This glandular inflammation may end in resolution, but often it terminates in suppurative or diffused necrosis. It may give rise to extensive gangrene of the tonsils and adjacent soft parts;

sometimes it is followed by extensive abscesses and destruction of the cellular tissue about the neck; the skin in the region may slough, and not infrequently fatal hemorrhage may result from the destruction of small vessels.

Diphtheria is so often a complication of scarlatina anginosa, that it has been assumed that there is some necessary relation between the two diseases. Yet diphtheria is as frequently met with in the mildest as in the severest types of scarlatina, and occurs in every stage of the disease; often it is present during the period of incubation, so that the symptoms of the two diseases appear simultaneously. Again, it is met with during the period of convalescence. In some instances, scarlatina seems to complicate diphtheria.

In a mild form of scarlet fever, when the disease runs a regular course, the nasal mucous membrane is usually pale, and its secretion is not increased. When the disease is severe, the nasal mucous membrane becomes secondarily, never primarily, involved. This is the result of a catarrhal affection of the throat. It is a purulent catarrh of the posterior nares, which gradually extends to the anterior nares, and gives rise to a very troublesome form of coryza.

During the eruptive period of scarlatina, affections of the ear frequently occur in connection with those of the throat. Usually these have their seat in the middle ear. They are always tedious and may become chronic.

Next to the skin and mucous surfaces, the kidneys are the organs most frequently affected in this disease. There is no question but that, in a certain proportion of cases, recovery takes place without any kidney lesions; but these are the exceptions and not the rule. The usual, and by far the mildest affection of the kidneys in scarlatina is a catarrh of the uriniferous tubules marked by a more or less extensive epithelial desquamation. In some epidemics the scarlatina poison induces croupous inflammation of the uriniferous tubules instead of simple catarrh.

The tubules of the cortical substance of the kidneys are most extensively affected; the morbid processes commencing at the Malpighian tufts follow the course of the convoluted

tubules. If the tubules are only slightly affected there will be no symptoms except a slight albuminuria.

In well-marked scarlatinal nephritis, the epithelial cells of the uriniferous tubes will be found clouded, enlarged, and changed in shape and position, and frequently entirely destroyed, or they may entirely block up the tubules. Circumscribed inflammatory masses will be found scattered throughout the substance of the kidneys; these cause the kidneys to present the appearance of interstitial nephritis. Sometimes abscesses form in the kidneys. These kidney changes are rarely well marked before the second or third week of the disease, and usually terminate in complete recovery; they very rarely lead to chronic kidney disease.

The character and extent of these kidney changes varies in different epidemics. During some epidemics, the kidney changes are slight; during other epidemics almost every case, whether mild or severe, will be attended by extensive kidney lesions.

At the post-mortem examination of scarlet fever patients, you will always find more or less extensive congestion of the internal organs, such as congestion of the brain, liver, spleen, etc., but these congestions do not vary in character from those met with in other acute infectious diseases. It has been said that the visceral lesions of this disease do not essentially differ from those of typhus fever, that there is the same tendency to softening of the spleen and liver, and that the condition of the cerebral vessels in the two diseases is very similar. In both, the changes in the constituents of the blood are such as to diminish its coagulating power; in both, the mucous membrane of the stomach and intestines undergoes similar changes, the Peyerian patches will often be found presenting the "shaven-beard appearance."

When scarlet fever poison, which usually only induces changes in the skin, throat, and kidneys, excites inflammation in the joints, pleura, and pericardium, these latter must be regarded as complications; they do not belong to the regular history of the disease.

ETIOLOGY.—The *cause* of scarlet fever is a peculiar substance which is transferable from the sick to the healthy.

Scarlet fever is unquestionably a contagious disease. It has been claimed by some that it is only propagated by contagion; by others that sporadic cases do occasionally occur; but there is little doubt, if the history of every case of supposed spontaneous scarlet fever could be carefully taken, it would be found that at no place and at no time had there ever occurred a case of spontaneous origin.

The disease may be conveyed directly from the affected to the healthy by contact. It may also be conveyed for a few feet through the atmosphere, and also by clothing which has been thoroughly saturated with the scarlet fever poison; therefore it may be considered a portable disease.

Animals that have been around those sick with scarlet fever may convey it. I now recall one instance in which the scarlet fever poison was conveyed in this way. For a number of days a little poodle dog had been around children sick with scarlet fever, and in a single visit to the children of another family the disease was conveyed.

There has been considerable discussion as to whether the disease can or cannot be conveyed in milk. I do not say that this is impossible, but I do not think it probable that it is so conveyed.

The infection of scarlatina is not so certain as that of measles or small-pox. When one member of a family is sick with measles, usually every other member of that family who has not had measles will contract the disease; whereas, one member of a family may be sick with scarlet fever and every other member may escape.

I stated that some persons seem to have a certain idiosyncrasy, so that when they are brought in contact with the typhus fever poison they do not contract the disease; so certain persons may be brought in contact with the poison of scarlet fever and yet not contract the disease. The poison which they receive into the system has power to produce some of the symptoms, but has not power to fully develop the disease.

Scarlet fever can be communicated from one individual to another by inoculation. If you take some of the watery material or serum that can be obtained from the minute

vesicles occasionally seen upon the surface of the body in connection with the scarlet fever eruption, and introduce it into the body of an individual who has not had scarlet fever, it will develop the disease. It has been proposed to inoculate persons who have not had scarlet fever in the same manner as one would inoculate persons who have not had small-pox, and, by so doing, produce a modification of the disease. But it has been found by experiment that those who have been inoculated for scarlet fever have suffered *more severely* than those who contracted the disease by any of the common methods of contagion.

There is no question but that the scarlet fever poison can also be introduced into the system through the respired air, but whether it can be taken into the system through the medium of food or fluids is still an unsettled question.

We are now brought to a question of great practical importance. If the disease can be conveyed by clothing, is it safe for a physician to visit patients sick with scarlet fever, and go from them directly to those who have not had the disease? Unquestionably, it is possible to so convey the disease, but in my own experience I know of no case where it has been so conveyed.

The clothing, in order to be sufficiently impregnated with the poison to render it a means of contagion, must be longer exposed than is the case when a physician makes a visit of ordinary length.

I do not hesitate to go directly from a patient who has had scarlet fever to one who has never had the disease.

While making my daily round of visits on scarlatina patients, I have frequently taken my own child, who has never had the disease, to ride with me, without fear of conveying to her the disease.

Unquestionably, nurses who have been with a scarlet fever patient for a number of days, and whose clothing has become filled with the poison, may carry the disease. Such persons should change their clothing before they go from the sick to the healthy.

With regard to the real nature of the scarlatina poison, the oft-repeated question comes to us, Is it a living organ-

ism or an impalpable poison? It is unnecessary to repeat what has been already said upon this point. The same arguments hold good in regard to this fever as in regard to the other fevers which we have been considering.

The period at which this disease is most infectious is probably the desquamative period, although some maintain that it is most infectious during the eruptive period. An individual is almost certain never to have a second attack.

The period of incubation varies from two to ten days, the average duration being from four to seven.

Age has a great influence on individual predisposition. The greatest susceptibility to the influence of the poison exists between the second and seventh years; it rapidly diminishes after the ninth year, so that adults, and especially the aged, have only a slight predisposition to the infection.

Scarlet fever may be endemic or epidemic. No reason can be assigned for the variations in type or severity of this disease. For years the type of fever which appears in a given locality will be exceedingly mild in character, and the cases will be mostly sporadic, when suddenly, without any assignable cause, a most malignant epidemic of the disease will prevail. Usually epidemics of scarlatina prevail in the autumn and spring.

SYMPTOMS.—The symptoms of scarlet fever vary with the type and with the severity of the fever. In moderately severe cases, before the appearance of the eruption, the patient will have more or less severe headache, pain in the back and limbs, and at first coldness of the surface. In some cases rigors will occur, and perhaps distinct chills. In children convulsions often occur. These ushering-in symptoms are immediately followed by a sensation of intense heat, with great acceleration of the pulse, which at this time often beats 120 or 130 per minute. There will also be nausea and vomiting, which symptoms are frequently the most persistent and distressing. Besides, there will be a rapid rise in temperature. It may reach 103° F. or 104° F., within a few hours. Within a period lasting from twelve to forty-eight hours, the average about thirty-six hours,

the eruption makes its appearance, and the fever increases. The elevation in temperature is accompanied by restlessness, a burning sensation, perhaps delirium; the nausea and vomiting become more urgent, and now the papillæ of the tongue become swollen, and the organ presents the appearance of a strawberry. It has been called the "strawberry tongue" of scarlet fever. This appearance is not commonly seen in the milder cases, but, as a rule, is present in all the severer cases. With the appearance of the eruption, all the symptoms, perhaps excepting the pain in the head, increase in severity. The urine, if it has been scanty, will now become more so, and may be nearly suppressed; if it has been sufficiently abundant, not unfrequently, as the eruption makes its appearance, the urine becomes scanty and high-colored.

In some cases the disease is so mild that there is but little disturbance, except that caused by the eruption. In other cases the disease is ushered in by violent nervous symptoms, such as delirium and coma, accompanied by extreme exhaustion, and the patient dies before the eruption makes its appearance. In other words, the patient dies during the period of invasion, from the overwhelming of the nervous system with the scarlet fever poison.

During the earlier stages of the disease the throat symptoms are quite characteristic. Adults and older children complain of a pricking sensation in the throat, and difficulty in deglutition; the tonsils, uvula, and posterior wall of the pharynx are red and œdematous, and from their appearance with the attendant symptoms, in most instances, you are very early able to decide that the case is one of commencing scarlatina. There are cases in which the throat symptoms are altogether absent at first, and do not come on until later in the disease.

We will now study in detail the symptoms which mark the development of this disease.

As I have already stated, the whole course of scarlet fever may conveniently be divided into three stages.

First, the stage of invasion, or the febrile stage.

Second, the stage of eruption.

Third, the stage of desquamation.

The duration of the stage of invasion varies in different cases according to the type of the disease. In most cases, it is from twelve to twenty-four hours; it may be four or five days. Usually the onset is marked by chilliness and slight rigor, followed by a rapid rise in temperature. The skin becomes dry, the face flushed, and the pulse accelerated. At the same time there is slight soreness of the throat, the face appears red and dry, the neck is stiff, and there is some tenderness about the joints. Vomiting and thirst are prominent symptoms. The tongue is red at its tip and edges, the papillæ are enlarged, and it presents the so-called strawberry appearance. Lassitude, pain in the head, aching of the limbs and restlessness are generally present. There may be some delirium at night.

Twenty-four hours after the commencement of the fever of invasion, an eruption makes its appearance, when the period of invasion is completed. The period of incubation, or the time which elapses between the exposure and the appearance of the eruption, varies. By some the eruption is said to appear as early as twenty-four hours after exposure, while others claim that one or two weeks may elapse after the exposure before the disease is developed, that the average time is six or seven days. You can make no definite statement in regard to the duration of the period between the exposure and the appearance of the eruption.

The eruption first makes its appearance upon the neck and upper portion of the chest, and is first seen as little red dots, varying in size from a line to a line and a half in diameter. These gradually coalesce and the eruption extends over the entire surface of the body, perhaps on the face, and lastly, it appears on the lower extremities. It presents its brightest appearance upon the evening of the fourth day.

On the morning of the fourth day, if you draw your finger across the surface, a clear, well-defined line will be made, which will remain for some time. The distinct white line which follows the finger is a point of some importance in distinguishing scarlet fever from roseola, a disease which

has an eruption closely resembling that of scarlet fever. In roseola, the well-defined white line produced by drawing the finger across the surface will be almost instantly displaced by the returning redness. It does not remain distinct as in scarlatina. The eruption remains visible six or seven days. Usually, it begins to fade upon the fourth day, and by the sixth day it has entirely disappeared, and desquamation has commenced. The period of desquamation lasts about two weeks, during which time there is the greatest danger of communicating the disease. At the end of that period, if no complication occurs, the patient is well. The fine scales which are so abundantly thrown off contain the specific poison, and they are so delicate that they are blown about with every breath, and carried in every current of air, and are in the most favorable condition to be taken into the system in the respired air.

Some have maintained that the contagious period in this disease does not occur until the period of desquamation. This statement is not sustained by clinical facts. The amount of the desquamation depends upon the intensity of the eruption. The skin has a dry feel before desquamation commences. Where the skin is thin, the epidermis comes off in thin scales. Where the skin is thick, as on the palms of the hands and soles of the feet, it peels off in extensive patches. With the desquamation, the fever subsides more or less rapidly.

LECTURE XXVII.

SCARLET FEVER.

Symptoms (continued).—Complications.—Sequelæ.

I WILL briefly repeat some things said at my last lecture in reference to the phenomena which attend the development of scarlet fever. Its symptoms may be divided into three stages: a stage of invasion, a stage of eruption, and a stage of desquamation. After a variable length of time from the exposure, varying from two to six days, the recipient of the scarlet fever poison begins to have chilly sensations, alternating with flashes of heat, rarely a distinct chill. Following this there is some soreness of the throat, headache, pain in the back and limbs; and the temperature rapidly rises, often in twelve hours reaching 104° F. With this rise in temperature there is an acceleration of the pulse, and not unfrequently very young children will be seized with convulsions, rapidly pass into a state of coma, and remain unconscious until the period of eruption. After the period of invasion has continued two or three days, a rash will appear, first upon the neck and chest; gradually it extends over the face and trunk, then is seen upon the extremities. This rash first appears as fine red dots; these dots form patches, which quickly coalesce.

After the second day of the eruption, if not before, the entire surface will present an uniform redness, the color varying with the severity of the disease. In the milder cases you will have a bright rose-red eruption or rash, while in

the severer types the eruption will assume an appearance resembling the deep-red color of the boiled lobster. The darker the eruption, the more severe the form of the disease and the greater the danger. When the eruption is fully developed you will notice that the surface is somewhat elevated, the parts present a swollen appearance, the vessels of the skin seem to be congested, and there will be soreness of the throat more marked than in the febrile stage. Usually, vomiting is present at the commencement of the disease, but becomes more severe and a more marked symptom as the stage of eruption is ushered in ; if not present at the commencement it is certain to make its appearance with the appearance of the eruption. The vomiting is peculiar, not on account of the matters ejected, but the *act* of vomiting is projectile in character. In scarlatina the condition of the throat depends upon the severity of the disease. In some cases there is simply a blush of redness over the posterior portion of the pharynx and uvula and anterior pillars of the soft palate. In other cases you will notice a general tumefaction of all the soft parts of the throat which can be seen, and the tonsils will be the seat of a more or less intense parenchymatous inflammation, which gives rise to a swelling that encroaches more or less upon the pharynx. Again, you will have ulcerative pharyngitis, as it is termed, or upon the surface of the enlarged tonsils and swollen mucous membrane of the pharynx you may have an exudation, which hereafter will be more fully described.

In the ordinary form of scarlatina, such as I am now describing, when it runs its regular course you will not have much swelling of the glands about the neck, nor very much tumefaction of the soft tissue in the pharynx.

The eruption will reach the maximum of development upon the fourth day, and will remain visible six days. Generally during this time the temperature continues to rise until perhaps it has reached 106° F. or 107° F. In the meantime the pulse may increase to 120, or even 140, or perhaps 150 beats per minute, and not unfrequently there is some delirium during this stage ; there may be also more or less stupor. There is an intense itching and burning upon

the surface, and an intense restlessness depending upon the congestion of the cutaneous covering of the body.

Upon the eighth day of the eruption you will notice that the temperature begins to decline, and at the same time it can be seen that the eruption has faded in a marked degree over the parts where it first made its appearance, especially about the neck. This fading of the eruption goes on rapidly, so that by the end of the eighth, certainly early on the ninth day, sometimes as early as the sixth day, there is no longer any eruption visible on the surface of the body.

With the disappearance of the rash, desquamation commences, and with this there will be a still more marked fall in temperature and diminished frequency of the pulse. All the febrile symptoms disappear, all the throat symptoms subside, there is no longer any difficulty in deglutition, there is no more pain in the throat, no more swelling of the external glands, if previously it had existed. The desquamation continues for from fifteen to sixteen days, after which time the patient is in a state of convalescence.

The entire period occupied by a case of scarlet fever when it runs its regular course is from two to three weeks.

Having given you a description of the development of an ordinary case of scarlet fever, I must state to you that this disease is liable to *irregularities* in its development and course, and to these it is important that I should direct your attention.

It is claimed by some that these irregularities depend upon the organ or set of organs primarily affected by the scarlet fever poison. They are rather due to some peculiarity in the type of the disease, to the degree of poisoning, and in some instances to the particular set of organs that are involved in the different epidemics.

In some epidemics you will see even milder forms of the disease than I have yet described. The attack may be so mild, and there may be so little fever, that if the eruption was not present, you would not be able to recognize the scarlet fever; and even that may be so slight that the stage of eruption and the stage of desquamation may pass unnoticed, and you may find yourself scarcely able to decide

whether the patient has, or has not, had an attack of scarlet fever

The most frequent irregularity in the manifestation of the disease is noticed in that class of cases where we have complications resulting from the overwhelming of the cerebro-spinal system with the scarlatina poison. This is due to some peculiarity of the poison, and is characteristic of certain epidemics.

In a large number of cases in the febrile stage, especially in young children, convulsions may occur, but they do not depend upon the peculiarity to which I refer.

In the class of cases to which reference has been made, where complications arise from the overwhelming of the cerebro-spinal system with the scarlatina poison, from the very onset of the disease there seems to be a tendency to stupor and delirium, a peculiar restlessness, an apparent wandering, a picking at the bed-clothes, accompanied by a peculiarity in the appearance of the eruption, which may cause it to assume the boiled-lobster appearance, or even a darker hue. The eruption is slow in its development, and there is not that uniform redness over the entire body that is seen in ordinary cases; it appears in patches, and with it there is exhibited a tendency to blueness of the finger-ends, indicating that there is acting upon the nervous system a poison which possesses the power of very greatly lowering the vitality of the patient.

There is a class of cases in which there is not much swelling of the throat, nor is the pulse more frequent than 130 or 140 per minute, but during the second day of the eruption the temperature ranges very high, reaching 107° F., or 108° F. Under such circumstances the disturbance of the nervous system is due to the high temperature which may have been present for two or three days; these disturbances may be prevented if the temperature is not allowed to rise above 103° F. or 104° F.

Again, in cases where there is marked swelling of the throat, and a general infiltration of the tissues and glands of the neck, the development of the nervous phenomena is due to an interference with the return circulation. The

condition which gives rise to the cerebral symptoms is one of mechanical cerebral congestion, if I may use the term in this connection.

There is still another class of cases in which the marked nervous phenomena appear still later in the progress of the disease. Under such circumstances they often indicate a typhoid condition. This typhoid condition is not induced nor are the nervous phenomena developed on account of the peculiar effect produced upon the nerve centres by the scarlet fever poison, nor are they due to the effects produced by a high temperature, nor by an interference with the return circulation, but they are due to septic poisoning, a poisoning entirely different from scarlet fever poisoning. The nervous phenomena develop after the eruption. During the developing period, you may have noticed a peculiar ichorous discharge from the nostrils, and frequently you hear it said that the patient has become re-poisoned by scarlet fever poison, but this is not the case: he has become re-poisoned by the septic element of these discharges.

During the period of desquamation you may have the nervous system involved, in consequence of the presence of uramic poisoning.

The mere terms, *scarlatina simplex*, *scarlatina anginosa*, and *scarlatina maligna*, do not indicate all that may be included under each division. You must remember that *scarlatina maligna* is that form of the disease in which the cerebro-spinal system becomes early involved, in consequence of some peculiarity of the scarlet fever poison; or it becomes involved while the eruption is being developed, and depends upon high temperature; or it becomes involved in connection with extreme swelling of the tissue of the neck, giving rise to interference of the return cerebral circulation, or in consequence of a septic or uramic element. What the changes are that produce these nervous phenomena, when high temperature is present, is still an unsettled question.

Again, scarlet fever may run an irregular course in those cases in which there is present an extensive infiltration of the tissue of the neck, with inflammatory products, swell-

ing of the glands, and extensive suppuration. Not infrequently these cases terminate fatally; doubtless in some cases the extensive suppuration in the areolar tissue about the neck produces this result, and in other cases it is produced by the interference with respiration caused by enlargement of the gland and swelling of the tissues of the neck. In these cases there is a certain amount of danger from œdema glottidis, the consequence of extension of the inflammation from the adjacent tissues.

There are cases in which the eruption is not very well marked; the patient passes safely through the stage of eruption, and the stage of desquamation is fully established; but, instead of making a good recovery from this point, immense abscesses are rapidly developed in the cervical region, blood-changes begin to manifest themselves—such changes as allow of the occurrence of hemorrhages—and the patient passes into a typhoid condition, with hemorrhages occurring from the nose, mouth, intestines, etc., and death ensues. Such a result is produced by the peculiar action of the septic poison developed during the suppurative process.

I have already referred to a scarlatinal coryza, in which the discharge contains elements capable of producing septic poisoning. I have come to regard this coryza as an unfavorable symptom. The clear serum which runs over the lip never causes death; but the fact that it sometimes produces excoriation and ulceration of the tissues with which it comes in contact, indicates that there are nasal and pharyngeal changes which may destroy life; especially is this the case in young children.

Sloughing ulcers sometimes develop in the mouth and throat; and, when they do occur, the patient is said to have ulcerative stomatitis; but these ulcerations are really due to a peculiarity of the scarlatina poison. Under such circumstances, your patient may go on through the period of eruption, enter the stage of desquamation, and then rapidly sink and die, with symptoms similar to those which attend diphtheria. Although the odor of the breath may very closely resemble that noticed in some cases of diph-

theria, there is no diphtheritic exudation present. When diphtheria does occur, it is developed as a complication or sequela; it does not belong to the regular history of scarlatina, and is an entirely different disease, depending upon an entirely different poison, which makes its appearance after the scarlet fever poison has spent itself. Remember that scarlatina and diphtheria are distinct diseases, and cannot be developed the one from the other, and that the condition I have been describing, which resembles diphtheria, is simply a scarlatinal coryza which indicates the existence of sloughing pharyngitis.

Scarlatina may also be made to run an irregular course by the development of inflammation of the internal ear. This inflammation extends from the throat up the Eustachian tube, involves the middle ear, and gives rise to a train of symptoms, such as intense pain, delirium, and rolling of the head, all of which suggest the presence of acute meningitis. I recall several instances in which the diagnosis of acute meningitis was made, where from the after history of the case there was no question but that the symptoms were due to such an inflammation of the middle and internal ear. When such an inflammation occurs, you should be prepared to relieve your patient. The method of procedure for the relief of this condition you will learn from lectures in another department of medicine.

All these differing conditions I have been describing are usually spoken of as complications of scarlet fever, but I believe them to be nothing more than a part of the regular history of the disease. We find the same thing true in regard to many other diseases.

COMPLICATIONS AND SEQUELE.—I come now to speak of those conditions which may be regarded as the sequelæ or complications of scarlatina. The most common sequela is anasarca. The anasarca of scarlatina usually appears at the time the patient is convalescing, during the period of desquamation, or just as desquamation is being completed. It has been commonly believed by the profession that anasarca is due to some exposure to the influence of cold during this period. It is quite possible that the changes in the

kidney which give rise to the anasarca may sometimes be produced by the influence of cold, and undoubtedly anasarca is occasionally developed in this manner, but in the majority of cases it is due to some peculiarity in the scarlet fever poison, or to some peculiar atmospherical condition.

During some years anasarca is a very common sequela of scarlet fever; while during other years we have equally severe cases of the disease, and yet scarcely a case of anasarca is seen. While we recognize the fact that it is possible for kidney lesions to be developed which shall give rise to anasarca in consequence of exposure to cold, it is also of importance that we recognize the fact that the lesions and the anasarca may be developed independent of such exposure. The anasarca first shows itself on the face, and from the face it extends over the entire body, and if it becomes general you will usually have more or less ascites developed. In most cases, at the time or previous to the occurrence of the anasarca, you will have certain premonitory symptoms, and it is of great importance that you should be familiar with these symptoms, and be on the watch for their appearance. For two or three days previous to their development a certain restlessness will be noticed, with nausea and vomiting. These symptoms are almost universally present.

The nausea and vomiting so commonly present during the earlier periods of the disease have subsided, and now, during the period of desquamation or perhaps after it has been completed, the vomiting returns. The patient has some pain in the head, has loss of appetite, is annoyed by the light, does not sleep well, and the temperature is raised perhaps two or three degrees. When your patient complains in this manner during the desquamative stage of scarlet fever, your suspicions should be aroused, and if you have not already examined his urine you should do so at once. It will usually be found scanty and high-colored, will contain albumen and casts of the exudative variety, and perhaps blood-casts. Occasionally, epithelial casts are found; usually, however, these casts are not seen until later during the disease. If you have made previous examinations of the urine before the development of these symp-

toms you may have found renal epithelium, which are usually found in any severe case of scarlet fever; but now there are present casts which indicate the existence of an active inflammatory process in the uriniferous tubules. It is not the epithelial desquamation of the tubules, which occurs in connection with the desquamation which is taking place over the entire surface of the body; but it is a distinct sequela of the disease, which shows itself in the form of a tubular nephritis. It is possible to have a parenchymatous nephritis developed in consequence of exposure to cold during this stage of scarlet fever, but this nephritis is due to the direct effect of a poison which is acting upon the secreting portion of the kidneys.

After the anasarca has been present two or three days, if the case is to have a favorable termination, the anasarca will begin to decline, will be less and less marked about the face and feet, the tendency to stupor which has accompanied it will begin to disappear; and as the dropsy subsides, and the patient is not so lethargic, the appetite begins to return, the urine increases in quantity, the albumen diminishes, the casts disappear, and convalescence is fully established. Anasarca may have been developed, all the symptoms have disappeared, and the patient have recovered within two weeks from the commencement of the attack. Such anasarca is due to a simple catarrhal inflammation of the uriniferous tubules, and as complete recovery may take place as after an ordinary catarrhal inflammation affecting the bronchial tubes.

If, however, after the anasarca is developed, the case is to go on to an unfavorable termination, the anasarca instead of diminishing will increase, the face will become more and more puffy, the legs more and more œdematous, the abdomen more and more distended, the pulse more and more frequent and feeble, the temperature more and more elevated, until a condition of coma is finally reached, which condition is sometimes preceded by convulsions, and followed by death.

I have given you a brief outline of the usual course of a case of scarlatinal nephritis, whether it goes on to recovery or to an unfavorable termination.

It is possible for bronchitis or pneumonia to occur as a complication of scarlet fever, but they are of rare occurrence. As I have already stated anasarca is the most common sequela, and if you will remember when and why it appears you will rarely fail to recognize its occurrence.

Another sequela of scarlatina is *inflammation of the serous membranes*. The serous membrane most liable to be involved is the endocardium, and this inflammation may pass unrecognized unless you are on the watch for its occurrence, for there may be no rational symptoms present. Endocarditis, when it does occur, is liable to be *ulcerative* in character. As the result of such ulcerative endocarditis you may have septic symptoms developed, or embolism occurring in consequence of the removal of a portion of material from the ulcerated valve, and a subsequent plugging up of an arterial twig in some distant part of the body. If a portion is removed and carried by the circulation into the brain, and has been lodged in one of the cerebral vessels, it will give rise to sudden coma, and unless you have been very closely watching your patient you may be at a loss to account for the sudden development of the embolic symptoms in a patient who seemed to be doing well.

If the endocarditis is not of the ulcerative variety, the patient apparently recovers and you discharge him as cured of his scarlet fever. Two or three months after his discharge, he comes back to you complaining of shortness of breath, and probably you will suspect and search for chronic kidney disease and find no evidence of its existence, but you will find the signs of chronic endocarditis, the result of the acute endocarditis, which you had failed to recognize.

Inflammation of the pericardium may occur as a complication of scarlet fever, but it does so much less frequently than inflammation of the endocardium. Inflammation of the pleura, and occasionally inflammation of the peritoneum is met with as a sequela of this disease. I have seen death caused by an acute peritonitis which occurred as a sequela to scarlet fever, but if peritonitis does occur it is much more likely to be subacute in character. It is possible to have peritonitis developed as a sequela to scarlet fever and to

be entirely recovered from. I have had two patients recover who had ascites, the result of subacute peritonitis as a sequela of scarlet fever.

Rheumatism may be developed during the desquamative period of scarlet fever. Under such circumstances it assumes the ordinary appearances of inflammatory rheumatism. Quite rapidly it invades one joint after another, the joints become red, swollen, and painful, the temperature rises, and the pulse becomes accelerated; but the attack is of short duration, usually does not last more than four or five days. It is not a serious sequela, and complete recovery usually occurs within ten or fourteen days from the commencement of the attack.

Suppurative inflammation of the joints sometimes occurs as a sequela of scarlet fever. I have seen cases in which suppuration of the knee-joint occurred after convalescence had been fully established, and all the phenomena of an ordinary attack of suppurative synovitis were presented. One case under my care terminated in ankylosis. Such suppurative inflammation is not of very frequent occurrence, but it is well you should be aware of the possibility of such a sequela.

Another serious complication of scarlet fever is diphtheria. It may occur at any period of the fever, usually it occurs during the period of desquamation. There is developed the characteristic exudation of the disease, with the attendant depression noticed in a case of diphtheria developed independently of scarlet fever.

It differs in no respect from primary diphtheria, except in the rapidity of its development and in its fatality. In scarlet fever there is no more serious complication. When I observe a diphtheritic patch in the throat of a scarlet fever patient, from that time I regard the case as hopeless. Usually it appears quite suddenly, and perhaps does not occur more frequently in those who have a severe form of the disease than in those who have a mild scarlet fever.

LECTURE XXVIII.

SCARLET FEVER.

Differential Diagnosis.—Prognosis.—Treatment.

IN the history of scarlet fever we have now come to its *differential diagnosis*.

DIFFERENTIAL DIAGNOSIS.—The diagnosis of scarlet fever is usually not difficult after the eruption has made its appearance, for, in well-marked cases, that alone will readily distinguish it from the other eruptive fevers. At the very onset of the eruption, and in *irregular* cases sometimes the differential diagnosis is difficult. The eruptive diseases which are most liable to be mistaken for scarlet fever are *measles*, *small-pox*, *roseola*, and an *erythema* which sometimes appears in surgical cases. In all doubtful cases a careful study of the history of the patient is necessary before making your diagnosis.

In measles the appearance of the eruption is preceded by a cough and coryza. These symptoms are never present in the ushering-in stage of scarlatina. Besides, the eruption of measles first appears on the face, whereas the eruption of scarlet fever first makes its appearance upon the neck and chest. After these diseases are once fully developed, the course of the one so differs from that of the other that there will rarely be any chance for doubt after the first week of the disease. The minute punctate appearance of the scarlatina eruption before it becomes confluent is an important element in its diagnosis. Although the eruption of *confluent variola*, for the first twenty-four hours, may sometimes

resemble that of scarlatina, yet the development of the first vesicle settles the question.

The appearance of *erythema* bears a closer resemblance to a perfectly developed scarlatina eruption than does any other eruptive disease. It is not, however, present on the extremities, neck, and portions of the trunk, and spreads in a very irregular manner; whereas in scarlatina such is not the case. But if, on account of the scantiness of the scarlatina eruption, any doubt arises as to the nature of the eruption, remember that in scarlatina the throat symptoms are rarely absent, that usually the tongue presents the strawberry appearance, and that at an early period there is usually some swelling of the cervical glands. In those cases in which, during the early part of the disease, it is impossible to make a differential diagnosis, when the period of desquamation is reached the diagnosis will be readily made.

The differential diagnosis between *roseola* and a very mild form of scarlatina is sometimes attended with great difficulty. If scarlatina is prevailing, and a child has an eruption which lasts for two or three days, then disappears, and is not followed by desquamation, you very naturally come to the conclusion that the case is one of scarlatina; and yet the sequela proves that the case was one of *roseola*. Such a form of *roseola* sometimes prevails epidemically, and attacks the children in a certain locality, whether they have or have not had scarlatina. Under such circumstances, adults and children are said to have had a second attack of scarlet fever.

In making a differential diagnosis between this form of *roseola* and scarlatina you must be guided by the duration of the eruption and by the character of the throat symptoms. In scarlatina the posterior part of the pharynx is affected, while in *roseola* the redness is confined to the anterior portion; besides, the throat affection in *roseola* is much milder than in scarlatina.

One can hardly mistake erysipelas for scarlatina, for erysipelas commences at one point and from that point gradually extends; there is also marked œdema of the con-

nective tissue, and there is a very marked difference in the constitutional symptoms of the two diseases.

There are malignant cases of scarlet fever in which no eruption appears; they prove rapidly fatal. In such cases, you must be guided in your differential diagnosis by the fact that an epidemic of scarlet fever is prevailing (which is usually the case), by the rapid development of the disease, by the very high range of temperature, and by the very grave nervous phenomena; all of which can only be accounted for on the ground that your patient is overwhelmed by some very active blood-poisoning.

In no other infectious disease do we have such violent symptoms, nor does death take place in so short a time.

In this class of cases you should frequently examine the entire surface of the body, for the eruption is sometimes very transient, perhaps appearing only for a few hours on the neck or extremities. It is sometimes difficult to draw the line of distinction between scarlatina without an eruption, with swelling of the cervical glands and ulceration of the throat, and diphtheria. If a patient has swelling of the cervical glands and well-marked febrile symptoms, which have come on gradually, that is, have been two or three days developing, and yet no scarlatina eruption has appeared, but a gangrenous ulceration has developed involving the tonsils, the posterior wall of the pharynx, and the anterior pillar of the soft palate, if scarlet fever is prevailing in the locality it is very difficult to decide between it and diphtheria.

There can be no doubt but that scarlatina poison may excite a tubular nephritis without an eruption appearing upon the surface of the body, or without any of the other ordinary symptoms of scarlatina.

PROGNOSIS.—The prognosis in scarlet fever is always uncertain. It will be influenced more by the character of the prevailing epidemic than by any other circumstance.

According to statistics, the rate of mortality ranges from one death in five to one in twenty. Some epidemics are very mild. During one epidemic, in one month, I treated fifty cases of scarlet fever, with only two deaths. During the

same month of the following year, I treated twenty cases with seven deaths. In making your prognosis you must always take into account the type of the prevailing disease. Even when the disease is mild in character, and is running a perfectly regular course, dangerous symptoms may suddenly arise without any assignable cause.

The conditions of a favorable prognosis are as follows : when the eruption appears within forty-eight hours from the commencement of the attack, and rapidly completes its course, reaching its maximum on the second day ; when the throat symptoms are mild, little difficulty being experienced in swallowing ; when the cervical glands are but slightly enlarged ; when the temperature does not rise higher than 104° F., and the pulse beats only 120 per minute ; when the cerebral symptoms are not severe, and are of short duration ; and when the disappearance of the eruption is attended by a steady decline in temperature. Even if there is a slight affection of the joints and a moderately severe nephritis during the period of desquamation, a favorable termination may be predicted. The nephritic symptoms will almost always entirely disappear during the third or fourth week.

The conditions for an unfavorable prognosis are : when the disease pursues an irregular course ; when the temperature rises above 105° F., with dyspnoea and extreme frequency of the pulse ; when symptoms of collapse come on, attended by a cold surface and a small pulse ; when the eruption assumes a livid hue, and there are abundant hemorrhages in the skin ; when ulcerative pharyngitis is present, especially when it extends to the nasal passages, accompanied by copious coryza and infiltration of the glands and tissues of the neck ; when severe nervous symptoms are developed with typhoid symptoms : when there is persistent and long-continued vomiting, with diarrhoea coming on at the commencement of the attack ; when the nephritic symptoms are early present, and there is general dropsy, excessive hæmaturia, or almost complete suppression of urine, with high temperature.

The occurrence of any of the more serious complications to which I have already referred, such as pneumonia, diph-

theria, pericarditis, œdema glottidis, etc., always renders the prognosis bad.

Before making your prognosis, decide whether the scarlet fever is regular or irregular in its course, and if irregular, what are the causes of the irregularity. By so doing, you will be greatly aided in making your prognosis. It is also important to determine your patient's power of resisting disease.

Favorable hygienic surroundings, good nursing, and well-directed medical treatment will greatly lessen the death-rate in scarlet fever epidemics, and these should be considered elements of prognosis. Patients with scarlet fever do better when left to themselves than when badly nursed, or when under the care of unskilful medical attendants.

Age is an important element of prognosis.

The period of greatest mortality is from infancy to five years of age. Beyond this period until adult life, the prognosis is decidedly better. In adults, the mortality is greatest in pregnant women, and those who are suffering from some organic disease, especially some disease of the heart or kidneys.

TREATMENT.—In connection with the treatment of this affection, the first question that presents itself relates to *prophylaxis* or *prevention*.

The *prophylaxis* of scarlet fever is a system of the strictest quarantine. The sick must be removed from the healthy. As in other exanthematous fevers, all useless articles of furniture must be removed from the sick-room. Fresh air renders the contagion of scarlet fever less powerful; therefore, free ventilation is of the utmost importance. All the clothes and excretions of the patient should be disinfected in the same manner as in typhoid fever. To prevent the dissemination of the dusty particles of the desquamating epidermis, during the period of desquamation the surface of the body should be frequently sponged, and after each sponging the surface should be rubbed with olive oil.

Those convalescing from this disease should not be allowed to leave their apartment until desquamation is completed, which usually requires at least three weeks after the com-

mencement of the period of desquamation. The sick-room and everything which has been used about the patient should be thoroughly disinfected, and the windows and doors of the apartment should be allowed to remain open for a long time before it is again occupied.

To prevent the spread of the disease, nurses and attendants upon the sick should not be allowed to have any intercourse with the healthy until the period of desquamation is passed, and after that time not until there has been thorough cleaning and disinfecting. It is doubtful whether the funerals of those dying of scarlet fever should be public.

There is no known prophylactic treatment, except isolation, and a thorough disinfection of everything contaminated by the contagion.

A theory has been advanced that belladonna has power to prevent the development of this disease in those who have been exposed to its contagious influence. This drug has been very extensively administered in order to test its effects as a preventive in scarlet fever.

After having carefully examined the subject, both in its literature and clinically, I am convinced that belladonna has no power to prevent the development or mitigate the severity of the fever in those who have been exposed to its infection. As I have already said, fresh air is the only agent of which we have any knowledge, which can render the contagious influence of this fever less powerful.

MEDICINAL TREATMENT.—The medicinal treatment of scarlet fever is almost entirely expectant. It must be remembered that it is a disease which cannot be aborted, and which, if left to its natural course, tends to recovery if the fever and the local symptoms remain within certain bounds. It has certain stages to pass through, and you cannot safely interfere with its regular course. Your province is to stand by and watch, and, so far as possible, guard against complications; if they occur you are able to afford a certain amount of relief.

There are certain details which it is important to attend to. The bed and body linen should be frequently changed. As soon as the period of desquamation has been reached the

patient should have a warm bath once or twice during the day, the surface of the body being well washed with carbolized soap. The baths hasten the process of desquamation and aid in bringing the skin into a healthy condition as rapidly as possible; the kidneys will also be relieved, and you may prevent serious lesions from these organs. Such general means as are applicable in the treatment of all fevers may be employed. If the temperature of the patient rises above 103° F., certainly if it rises above 104° F., it is important that some measures be resorted to for its reduction. The temperature should never be allowed to remain at 104° F. longer than twenty-four hours.

The means which are to be employed to accomplish this reduction are the antipyretic measures already referred to, such as the application of cold to the surface by means of sponging and baths, and the administration of large doses of quinine.

There is a strong prejudice against the application of cold to the surface of the body in scarlet fever. I am by no means certain that cold baths are always safe, or that in all cases the application of cold to the surface is judicious treatment.

At the present day, we are told that the kidneys will be most readily relieved of the scarlet fever poison when cold is used for the purpose of reducing the temperature, and that we should make use of this agent rather than permit the case to go on without effecting such a reduction.

It is claimed that when the temperature of a patient is kept below 103° F., scarlatina nephritis rarely occurs. This statement is not sustained by facts; it has been found that kidney complications are as extensive in the cases where cold is employed as in those cases where the temperature ranges higher and cold to the surface is not employed.

We should be governed by the same rules in the application of cold to the surface in scarlet fever as govern us in the treatment of typhus and typhoid fevers.

With regard to the use of quinine as an antipyretic, I need add nothing to what has already been said in connection with its antipyretic power in the treatment of other

fevers. It has the same power of reducing temperature in scarlet fever that it has in typhoid fever.

Unless the temperature in a case of scarlet fever ranges above 105° F., do not apply cold to the surface, nor give quinine in antipyretic doses. With such a temperature there will probably be delirium, but it must be regarded as one of the phenomena of the disease, requiring no special treatment. If the temperature rises above 105° F., perhaps reaches 106° F. or 107° F., and the patient manifests the nervous phenomena which have been referred to, such as restlessness, tossing, blueness of the surface, tendency to coma, etc., your duty is to reduce the temperature either by the application of cold to the surface or by the administration of one or two antipyretic doses of quinine. In all cases, let the patient be sponged frequently with tepid water, and if there is intense burning of the surface, add a saline to the water. Sponging in this manner will give the patient very great comfort. Some have advised that the surface be anointed with oil for the relief of the burning. My own experience has led me to rely upon simple tepid saline water. I have found that it gives patients greater relief, is more easily applied, and in its use is more agreeable than any of the substances which have been used for this purpose. I have not found that the application of oil to the surface has any effect in controlling the temperature, nor does it seem to have any effect on the process of desquamation, and as soon as desquamation commences, the process should be assisted by frequent washings with soap and water. For the throat complications, which will give you more or less trouble in all severe cases, especially when there is much enlargement of the glands at the angle of the jaw, causing difficulty in swallowing, leeches were formerly employed, but their use has now been almost entirely abandoned. The vitality of the patient is lessened by their use, and on this account they are contra-indicated. Of all the remedies which I have employed for the relief of throat complications, I think cold carbonic acid water the best. Whether it does more than afford relief, I am not able to say, but I am certain that cold carbonic acid water

or pieces of ice held in the mouth, and brought as much as possible in contact with the swollen mucous membrane of the throat, when used early, afford most marked relief. In the advanced stages of the disease, where there is great infiltration of the glands and tissues about the neck, cold applications do not afford the same relief as when they are used in the early stage; then cloths wrung out in tepid water and applied to the surface seem to be of service. During this stage, hot applications are generally much more agreeable to the patient. You may cover the hot cloths with oil-silk. These applications will not hasten the suppurative process, unless suppuration is already established. While using hot applications externally, warm water gargles and steam inhalations may be used internally. Of these methods of treating throat affections, adopt the one which seems to you to be the most rational plan of treatment. In scarlet fever I favor the use of hot rather than cold applications. Whichever you use, use it to the exclusion of the other; either cold internally and externally, or heat internally and externally.

There are different opinions in regard to the action of heat and cold. Some claim that their action is the same.

The superficial and deep ulcers which are sometimes seen in the throat of scarlet fever patients can best be treated by spraying them with carbolic acid, muriated tincture of iron, chlorate of potash, tannic acid, or any of that class of remedies. Whatever remedy you may choose, it can be much more successfully applied by means of spray than by a camel's-hair brush or a probang. Such local remedies thus applied afford great relief. The pain from these ulcerations is sometimes very severe, and you will be obliged to resort to some measure for its relief. Bromide of potassium, ether, and other anodyne applications in the form of spray may be made with satisfactory results.

In a certain class of cases, where there is marked disturbance of the nervous system, accompanied by great depression of the vital and feeble heart action, you will be obliged early to resort to the use of stimulants. It is not necessary to wait until a certain stage of the eruption or of the dis-

case is reached before commencing their administration. It may be necessary to resort to their use within twelve hours, or even within a less time, from the commencement of the attack. In some cases you will rely almost entirely on the beneficial effect that may be produced by the free and early administration of stimulants. The approach of kidney sequela in scarlet fever will be indicated by the development of those premonitory symptoms which precede the anasarca; and whenever such symptoms are developed, you should apply dry or wet cups, according to the condition of the patient, over the region of the kidneys, upon either side of the spine. Apply three or four cups on each side, and follow their application with hot fomentations over the kidneys. At the same time raise the temperature of the sick-room to 73° F. or 74° F., cover the body of the patient with flannel, administer hot-air or warm baths, and early commence the administration of diuretics. Of these, digitalis will act most favorably. If the anasarca does not disappear under the influence of the digitalis and the other means employed, calomel may be combined with the digitalis, and its use continued for a few days. Why the action of diuretics is increased by having a mercurial combined with them I am unable to say; but the fact is well established that, in certain cases—when the patient is going on from bad to worse, when the anasarca is increasing, the tendency to coma is becoming more and more marked, indicating an unfavorable termination to the case, and cups have been applied, hot baths, and diuretics employed with no satisfactory result—if small doses of calomel are combined with the diuretics, and their use continued for two or three days, the entire phase of the case may be changed.

In conjunction with the measures recommended, let the patient drink as freely as possible of water. If convulsions occur, or threatening symptoms indicating the approach of convulsions, are developed, you will be justified in resorting to the use of opium, either hypodermically or by the mouth. Under such circumstances the effect of opium is often most satisfactory. It not only arrests the convulsive tendencies, but produces the most profuse diaphoresis, and

aids in restoring the renal functions. With this class of patients I am confident that I have saved life by the timely use of opium. In my published articles on Bright's disease I have very fully discussed the subject, and given the reasons for its administration.

It is unnecessary for me to detain you with the special treatment of the different complications which I have stated as liable to occur in scarlet fever. The treatment of each complication will be indicated by the character and severity of the complication.

There are many other minor points in the management of this disease. I have given you an outline which I think will enable you to fully appreciate the general indications, and I must leave many of the details of treatment to your own study and experience.

LECTURE XXIX.

MEASLES.

Morbid Anatomy.—Etiology.—Symptoms.

WE now come to the study of another exanthematous fever, namely, *measles* or *rubeola*. This is of much more frequent occurrence than any of the fevers which have been engaging our attention. It is a disease from which few persons escape. It is essentially a disease of childhood, but it may occur at any age; it is, however, less liable to occur in young infants than in children after the period of dentition. A second attack is of rare occurrence. It is characterized by an eruption of red spots, accompanied by a catarrh of the mucous membrane of the air-passages, and a more or less severe fever. It may prevail as an epidemic or endemic disease, and not infrequently there are sporadic cases of measles.

MORBID ANATOMY.—The anatomical lesions of measles, with the exception of the eruption, are similar to those of small-pox and scarlatina. There are the same changes in the blood: it is dark-colored and fluid, poor in fibrin, and in severe cases shows a tendency to infiltrate the tissues. The number of red globules are diminished, and the white ones are increased. There is the same tendency to congestion of the internal organs. The spleen and liver are moderately enlarged. The mucous membrane of the nose, pharynx, larynx, and larger bronchi, as well as the conjunctiva, are more or less intensely congested, and present all the ana-

tomical changes of acute catarrh. In the majority of instances this catarrh is most severe just before and during the early period of the eruption; generally, it begins to disappear when the eruption has reached its height, and within two or three days entirely disappears. Where death has resulted from measles, in the majority of autopsies you will find evidences of capillary bronchitis, and not infrequently evidences of catarrhal pneumonia. Strictly speaking, these are not anatomical lesions of measles, but complications; they are, however, such frequent attendants of this disease, that they almost become a part of its history. Catarrhal affections of the respiratory organs are rather characteristic of the measles. The eruption of measles is papular; the papules first show themselves upon the face, especially upon the chin; gradually they extend to all parts of the body, until lastly they are seen upon the back of the hands. When the eruption is well developed the spots are slightly elevated, and have a diameter varying from one-tenth to one-twentieth of an inch; in form they are crescent-shaped, their margins are sharply defined, usually their color is of a bright red, sometimes shading off into blue. In most cases the spots are distinct and separated from each other by pale tracts of skin; they may become confluent, and thus give to the surface an uniform redness. When this occurs the surface presents an appearance similar to that seen in scarlatina. The earlier papule in each spot usually occupies the place of a hair-follicle. The spots disappear on pressure, but immediately return when the pressure is removed. Sometimes each spot contains several papules. The diversity in form and appearance of measles spots in different cases depends upon deviations in size, elevation, and grouping of the papules. When the spots assume a dark-red color, and do not disappear on pressure, capillary hemorrhages have taken place into the papules, and the eruption is called hemorrhagic. When the eruption is very abundant, little vesicles sometimes appear upon the papules, especially upon the trunk when there has been profuse perspiration. As soon as the spots have reached their maximum of development, their color

begins to fade; the fading is progressive, the centre of the spots longest retain their redness; the elevations subside with loss of color. In a varying time, from one to five days, the spots entirely disappear, leaving a yellowish or brownish stain. This staining is due to pigmentation of the skin, and is sometimes visible for two weeks. Exfoliation of the epidermis or desquamation takes place only upon the sides of the measles spots; it is never so extensive as in scarlet fever. The skin does not desquamate in layers, but in fine brown scales. It may commence before the redness of the eruption disappears, but it does not usually occur until the eruption has entirely faded. In most cases the period of desquamation is short, rarely lasting a week.

ETIOLOGY.—As regards the etiology of measles, experience teaches that it is essentially a contagious disease. So far as has yet been determined, it is only propagated by contagion. There are places, extensive districts, and countries thickly inhabited, where this disease has never prevailed. There is no authentic evidence that it ever *originated spontaneously*.

A few years ago, one of our own countrymen announced that he had found in decaying straw a peculiar growth or fungus which had the power of developing measles.

During the late war we frequently heard of "straw measles." When the surface of the body was brought in contact with a fungus found upon decaying straw, an eruption was developed. The eruption was not that of measles. It had no power of propagating itself, and could not be conveyed from one individual to another.

The question has often been asked, where is the poison of measles located? I answer, either in the mucous secretion, or in the exhalations from the body of the infected, so contaminating the air about the sick, that when persons who have not had the disease are brought within its influence, measles will be developed. It has been proved that the blood, the mucous secretions, and even the tears have the power of conveying the disease by inoculation. I suppose there is little question but that the disease can be conveyed in clothing, or, in other words, that it is a portable

disease. I regard the infection of measles as more tenacious, so to speak, than that of small-pox or scarlet fever. That is, a person not protected when exposed to measles is much more certain to contract the disease than is an unprotected person to contract small-pox or scarlet fever, the same circumstances surrounding the exposure. It is possible for the infection to be conveyed from one place to another in clothing and in fluids. I know of one instance in which it was brought to a family in cow's milk. The exact nature of this poison is still unknown.

It has been claimed that a certain cell has been found, a cell with a tail-like end, movable and colorless, which has the power of developing measles, but these statements have never been substantiated, and like the theory that the syphilitic cell was the active agent in the development of syphilis, this theory of development still lacks facts to sustain it.

The microscope has not as yet revealed the contagion of this disease. All that can be said with positiveness concerning its nature is, that there is an impalpable virus which may be conveyed from an affected to an unaffected person, and when received into the body of an individual who is not protected from the contagion by a previous attack, after a certain period, varying in length from eight to fourteen days, it produces the phenomena which characterize the disease. Some claim that the poison may remain sixteen days in the system before the phenomena of the disease are developed. One case is recorded in which the disease is said to have been developed fifty days after exposure.

This period is termed the "period of incubation," and its average duration is eight days. During this period the poison remains latent, giving its possessor no knowledge of its presence.

In most cases a slight exposure is sufficient to induce the disease; in some cases it is contracted only after prolonged exposure.

Susceptibility to this contagion is almost universal. All classes are equally subject to the infection. Second attacks are exceedingly rare.

The exact time in the course of the disease when measles is most infectious is not definitely determined. Statistics furnish almost absolute proof that it may infect throughout its entire course, in the precursory, eruptive, and desquamative stage.

SYMPTOMS.—Measles, like the other exanthematous fevers, if uncomplicated, runs a definite course. I shall describe the course of an uncomplicated case of ordinary severity.

As I have already stated, the *stage* of incubation is the latent period of the disease, without fever, and free from local symptoms.

Premontitory or precursory stage.—At the end of this period, or from eight to ten days after exposure, the patient begins to suffer from coryza, is languid, chilly, and exceedingly irritable. Occasionally, in young children, convulsions occur. The coryza and other catarrhal symptoms, at first, may or may not be accompanied by fever. Very soon they will be followed, if they are not accompanied, by a marked febrile movement. The eyes will be injected and watery, there will be a burning sensation and an aversion to light, and the eyelids will be red and tumefied. There is a constant, irritating, watery discharge from the nose, with frequent sneezing and pain over the frontal sinuses. Sore throat is complained of, and the voice is a little husky. Bronchial catarrh is indicated by uneasiness and constriction over the chest, with a frequent, dry, hoarse cough, hurried respiration, etc. The suffused, red appearance of the eyes is peculiar, and distinguishes measles from scarlet fever and other forms of eruptive fever. After the early symptoms have continued perhaps for twenty-four hours, perhaps no more than two or three hours, an initial fever will be developed, which, with the catarrhal symptoms, continues for about forty-eight hours; then the eruption makes its appearance.

Eruptive stage.—The eruption is first seen upon the face, then upon the neck, then upon the chest and over the body, afterwards upon the legs and arms, and lastly, upon the back of the hand. Usually it is about four days from the time of the appearance of the eruption upon the face be-

fore it has passed over the entire body, and it begins to fade from any one part about thirty-six hours from the time of its appearance upon that part; first, it begins to fade from the face, then the neck and chest, and finally from the back of the hands. If you closely examine the eruption it will be found composed of little fine red dots, which, after a little time, will be seen crowded together in patches of irregular shape. Usually these patches are crescentic in shape, and between them will be skin having its natural appearance. In this respect, the eruption differs from that of scarlet fever. In scarlet fever there is a uniform blush or redness, and when the eruption is present no portion of the skin remains unaffected.

The eruption of measles presents more of a papillary appearance upon the face than upon any other part of the body.

With the appearance of the eruption there is more or less swelling of the surface, itching and burning, and the color of the eruption will vary from a bright rose red to a dark mahogany hue. The difference in color depends upon the condition of the individual and the peculiarity of the type of the disease, rather than upon any change in the skin itself. As the eruption disappears it loses its bright red color, and becomes a yellowish-red, until, finally, nothing but a staining of the surface is left, then desquamation commences.

DESQUAMATIVE STAGE.—The desquamation which follows the eruption is not like the desquamation of scarlet fever, occurring in patches, but it occurs in very fine dust-like flakes, which may pass unobserved. The eruption reaches its height by the third day from the time of its appearance, and generally has disappeared by the end of the sixth day. As a rule, during the development of the eruption, the catarrhal symptoms and fever are increased in intensity; the patient will sneeze and cough, and frequently with such severity, and with such a coarse, grating, brassy tone, that it has received the name of "*iron cough*." It is not the cough of croup, there is no stridulous breathing accompanying it, but it is the result of an ordinary catarrhal laryn-

gitis, which causes the patient to cough perhaps for two or three days without expectoration, or any attempt at expectoration. During this period the pulse will range from 100 to 120 beats per minute, and in young children may reach 160 beats per minute. In the majority of cases, the temperature does not rise above 103° F., but it may rise as high as 106° F. or 107° F. As soon as the eruption begins to decline, a marked effect will be produced, and usually the temperature falls two or three degrees. As the decline in the eruption goes on, the temperature gradually falls, until, by the time the eruption has entirely disappeared, the patient will be fully convalescent.

Such is a brief description of the eruption, and the symptoms accompanying it, when measles runs its regular course. There are certain irregular modes of development which you will do well to remember.

We have different varieties of measles, if we may regard them as varieties.

We have, first, the regular form of measles, which we have just been considering, in which the disease runs a regular course, and the eruption has its regular stages of development. Then, when measles is prevailing in a locality, you will meet with cases in which all the catarrhal symptoms of the disease are present, without an eruption. You will also meet with cases in which there is an eruption closely resembling that of measles, with no catarrhal symptoms: from the appearance of the eruption, you will not be able to say whether the patient has or has not measles: if he has been exposed to the contagion of the disease you will be inclined to regard the case as one of measles, and yet if there are no catarrhal symptoms, but simply an eruption, I should hardly be willing to make such a diagnosis. There is a form of roseola which very closely resembles measles in every aspect of the disease, except the catarrhal symptoms.

There is an irregular form of measles which prevails epidemically, which is characterized by a tendency to ulceration of mucous surfaces. This form shows its peculiar tendency by the development of ulcers at the angle of the

mouth, within the nose, around the vulva, anus, etc. Sometimes these ulcers spread and so interfere with deglutition and respiration as to endanger life. The ulcerations are accompanied by great prostration of the vital powers and a tendency to gangrene. This irregular variety only occurs in those who are poorly nourished, live in badly ventilated houses, and are surrounded by unfavorable hygienic influences.

Again, there is another form of measles in which, at the very onset of the disease, there is a very high range of temperature. You will have no more severe catarrhal symptoms than in the ordinary forms—no more bronchitis; but there will be more fever and a higher range of temperature, the temperature perhaps ranging as high as 106° F. or 107° F. Associated with this elevation of temperature, there will be a restlessness, a dry tongue, and, very soon after the appearance of the dry tongue, a change in the color of the eruption, and it will assume a dusky, purplish hue. The eruption may present this peculiar appearance at the very commencement of its development. This type of measles is called "*black measles*." The color of the eruption simply shows that there have been extensive blood-changes. In most cases, quite probably, these changes have taken place prior to the development of the eruption. By some it has been claimed that there is at work a peculiar epidemic or endemic influence that gives rise to the peculiar type of the disease; but, as I have been brought in contact with it, it has seemed to me that it differed from the ordinary type only in the intensity of the fever. It is the high range of temperature which stamps it as a peculiar type of the disease; but, as soon as the eruption has made its appearance, although at first it may be of a bright red color, within a day or two it assumes the peculiar dusky black appearance which has given rise to the name it has received.

There is another irregular form of measles, in which the eruption is largely made up of petechial spots scattered over the surface of the body, which are due to a hemorrhagic diathesis. It is really a hemorrhagic form of measles,

and is a very unfavorable type of the disease. At first the eruption presents the same appearance as the ordinary eruption of measles; but, after the fever has continued a few days, it assumes a dark color, the patient becomes restless, the tongue dry, there may be vomiting and diarrhoea, and, if death occurs, at the post-mortem examination you will find that the anatomical lesions very closely resemble those found at the post-mortem examination of one who has died from typhoid fever, such as changes in the spleen and elevation of Peyer's patches. These cases are also known by the term "black measles." We have, then, two forms of black measles—one in which the eruption consists of petechial spots scattered over the surface, and dependent upon a hemorrhagic tendency; in the other form the eruption assumes a dark appearance, on account of changes which have occurred in the blood, the result of a very high degree of temperature at an early period of the attack.

I have thus briefly spoken to you of the most frequent irregularities in the course of this disease. There is always more or less danger connected with any of the more severe forms of irregular development. Although measles is usually not a disease of much severity, yet you must remember that, however mild the type may be, the disease is liable to be complicated, and the most frequent complications are to be found in the respiratory organs.

COMPLICATIONS.—Of these the most important is capillary bronchitis. You will rarely have a case of measles without more or less bronchial catarrh, but the bronchial catarrh which ordinarily attends it is not of much consequence. When, however, you find that the bronchitis is becoming capillary, you must recognize the fact that the patient is in great danger. Upon auscultation, if instead of loud, sonorous râles, which indicate that the catarrh is confined to the larger bronchial tubes, you have fine crackling sounds, accompanied by an entire loss of or an extremely feeble vesicular murmur, you may be certain that the catarrhal inflammation has extended into the finer bronchial tubes, and when, in connection with this disease, these are

invaded, you should remember that there is always great danger of the plugging up of the fine bronchial tubes. This will almost certainly be followed by a lobular collapse, and a subsequent development of lobular pneumonia.

A catarrhal pneumonia which complicates measles is always attended with great danger.

As a rule, it attacks both lower lobes at the same time, especially their dorsal aspect, while in the upper lobes only a few tubes are involved. This complication may occur at any time during the course of measles, but it is more liable to occur just after the eruptive stage. Its development always increases the fever in proportion to the extent of lung involved.

Desquamative nephritis may occur as a complication, but is not of as frequent occurrence as in scarlet fever. You will rarely have anasarca or the other attendants of scarlatinal nephritis.

Secondary meningitis not infrequently occurs as a complication in measles. When it does occur, it is developed during the period in which the eruption is disappearing. It is more likely to occur in this disease than in scarlet fever.

In connection with measles you will have what may be regarded as a sequela, a mild form of ophthalmia. This ophthalmia may considerably inconvenience the patient, and lead to permanent injury of the eyes. It is especially important that you should remember that it appears during the convalescing period, that it is a conjunctivitis, and usually entirely disappears if the eyes are frequently bathed with warm water and properly protected from the light.

Otorrhœa, or inflammation of the external ear, is another complication, or rather sequela of measles. It most commonly appears in those patients who have what is called a strumous diathesis, have phthisical parents, are themselves badly nourished, and have suffered from a severe form of measles. This otorrhœa is sometimes very obstinate, and if it yields to treatment, does so very tardily.

In adults, acute miliary tuberculosis not infrequently occurs as a sequela of measles. This is the unqualified statement of the books.

Within the past two years I have seen two cases of what, previous to death, seemed to be acute tuberculosis, and when the autopsy was made, throughout the lung substance here and there were little points or nodules which presented the usual appearance of miliary tubercles, but, when microscopically examined, they were found to be points of vesicular pneumonia. These two patients really died from pneumonia, and not from acute tuberculosis, although the lungs presented the appearances ordinarily seen in connection with acute tuberculosis.

The gross appearance of the lungs so closely resembles lungs that are the seat of acute tuberculosis, that it is difficult with the naked eye to distinguish the one from the other.

The mucous membrane of the intestinal canal may also become the seat of important complications in measles. A mild form of gastric catarrh is of quite frequent occurrence, but is rarely serious in character. Severe intestinal catarrhs, giving rise to troublesome diarrhœa and dysentery, are sometimes very serious complications, especially in very young and feeble children. Occasionally malignant epidemics of measles prevail, during which fatal results are chiefly due to intestinal catarrhs.

Diphtheria does not so frequently complicate measles as it does scarlet fever. It generally makes its appearance at the acme of the eruption, and when severe its occurrence is marked by a rapid rise in temperature. The symptoms of the diphtheria are the same as when it occurs as a primary disease. Inspection shows the diphtheritic exudation on the tonsils and pharynx, accompanied by all the attendant phenomena of ordinary diphtheria. Sometimes the diphtheritic exudation appears on the labia of the female, and on the genitals of the male. It must always be regarded as a serious complication.

Not unfrequently measles leaves the patient in a state of general ill-health. Especially is this the case in scrofulous and rachitic children.

LECTURE XXX.

MEASLES.

Differential Diagnosis.—Prognosis.—Treatment.—Roseola.—Miliary Fever.

WE will continue the history of measles, and this morning I invite your attention to its differential diagnosis.

DIFFERENTIAL DIAGNOSIS.—Ordinarily, when the eruption is well defined, the diagnosis of measles is not difficult. In some cases, however, the eruption presents an appearance which closely resembles that of the eruption of scarlet fever and roseola.

As I have already stated, in nearly every case of measles the catarrhal symptoms precede and accompany the precursory stage, and increase in severity during the period of eruption. The presence or absence of these catarrhal symptoms will enable you in the majority of cases to make a differential diagnosis.

It has been said that the line of distinction between measles and scarlet fever may be easily drawn; that if in scarlet fever you pass your finger-nail lightly over any portion of the surface of the body, a white line will remain, which will immediately again become red. Whereas in measles no mark will usually be left; but, if a white line does remain, the color produced is more permanent than in scarlet fever. In well-marked cases this appearance may settle the question of diagnosis, but in those cases in which the eruption of measles closely resembles that of scarlatina,

we are compelled to rely upon the presence or absence of catarrhal symptoms and the appearance of the throat. In children, the eruption of typhus fever very frequently closely resembles that of measles, but it does not appear upon the face, and is not accompanied by catarrhal symptoms. In typhus fever, quite frequently, nervous symptoms are present, such as delirium, prostration, and tendency to coma. Such symptoms are only met with in the hemorrhagic or typhoid variety of measles. Before the appearance of the eruption a careful examination of the mucous membrane of the pharynx will settle the question of diagnosis. In measles the mucous surface will be more or less intensely injected; in typhus fever it will not be so injected.

The differential diagnosis between measles and small-pox has already been considered. There will certainly be no difficulty in making a diagnosis, if you wait until the third day of the eruption: then the small-pox vesicle is formed. The same is true of varicella and other vesicular diseases.

The eruption of measles differs from that of roseola. In measles it is partially confluent, in roseola it is non-confluent. In roseola the mucous membrane of the fauces is not intensely injected. In measles the fever runs a characteristic course. If the temperature is normal, if the eruption on the trunk is of a bright red color, if the surface is smooth, and if catarrhal symptoms are absent, you may exclude measles. The non-contagious character of roseola is an important element of differential diagnosis.

It is hardly possible to mistake syphilitic *exanthemata* for measles, for there are certain glandular changes which attend the development of syphilitic eruptions which establish the diagnosis. In the early period of the disease, when coryza is a prominent symptom, before the appearance of the eruption, measles may be mistaken for an ordinary influenza.

PROGNOSIS.—The prognosis in uncomplicated measles is always good. Any irregularity in its development, and dentition in children, may render the prognosis unfavorable. In the hemorrhagic, in the ulcerative, and in the ty-

phoid variety, or black measles, as it is termed, the prognosis is grave. Measles occurring in pregnancy almost invariably prove fatal.

In severe cases, the deviations from the typical course of the disease which render the prognosis unfavorable are a temperature of 105° F. or 106° F., during the period of initiatory fever, a retardation or an irregularity in the appearance of the eruption at the beginning of the eruptive stage, and the occurrence of complications, especially bronchopneumonia, croupous laryngitis, and diphtheria.

Profuse hemorrhages from the mucous surfaces during any period of the fever, render the prognosis unfavorable.

The hygienic surroundings of the patient greatly influence the prognosis.

The prognosis also depends upon the age of the patient; the rate of mortality is much greater among adults than children. The character of the prevailing epidemic determines to a very great degree the prognosis.

When measles is developed in one who is suffering from a severe chronic disease, especially some organic disease of the lungs, the prognosis is unfavorable. The patient will not probably die during the active period of the measles, but the chronic pulmonary disease may terminate fatally from the effect produced by the sequelæ of measles. For instance, a person has evidences of consolidation about the apex of the lung, a condition which justifies a favorable prognosis; let measles be developed in this same person, and capillary bronchitis, terminating in a more or less extensive pneumonia, will probably occur, from which acute phthisis may be developed.

In measles, death rarely occurs during the first week of the disease; it usually takes place during the second week; if serious complications occur, it may take place later in the disease.

TREATMENT.—The prophylactic treatment of measles consists in isolating the affected person.

When the disease runs its regular course, the principal duty of the physician is to watch for and guard against the occurrence of pulmonary complications. As regards treat-

ment, all that is necessary is to place the patient in a large, well-ventilated room, with the temperature of 63° F. or 65° F. The diet should be milk. The room should be darkened, so that the congested conjunctivæ may not be exposed to light. If the patient complains of itching and burning of the surface, he may be frequently sponged with tepid water, this causes an alleviation of the itching and burning, and reduces the temperature. In an ordinary case this is all that will be required. Hot drinks or stimulants have no power to hasten the appearance of the eruption; the administration of the latter may be followed by very injurious results; convulsions may occur and death ensue.

In an ordinary case, stimulants should never be administered during the initiatory period of the fever, unless there is some special indication for their use, such as great prostration, or bronchial complication; then they may sometimes be used with benefit. Covering the patient with heavy clothing does not hasten the appearance of the eruption.

The greatest cleanliness should be observed; besides, there should be free ventilation, avoiding all draughts in the sick-room. If there is thirst, cold water may be freely taken in small quantities at a time.

If the case is severe, and the temperature rises to 103° F. or 104° F., it may be reduced by frequently sponging the surface with tepid or cold water. German writers recommend the cold bath in the treatment of measles. I should hesitate to place a patient with measles in a cold bath, on account of the great tendency in this disease to pulmonary complications.

Only a few days since I saw a child sick with measles, who had been treated with cold baths for the reduction of temperature. I found the physical evidences of extensive lobular pneumonias, which the attending physician said had been developed within the previous twenty-four hours, so that there was little doubt but that they were developed subsequent to the baths.

My own experience leads me in the treatment of measles to employ quinine as an antipyretic, in preference to cold to the surface, either by baths or packs.

You will recollect I stated that the post-pharyngeal catarrh is liable to extend into the larynx and bronchial tubes and give rise to bronchitis. One of the most important duties of the physician is to watch for the occurrence of this complication; he should frequently examine the chest, and when the bronchitis is found to have reached the capillary tubes, should immediately commence treatment for its relief. I have found the inhalation of steam to afford the greatest relief and best control the bronchial inflammation. As soon as I find that the larynx has become so involved as to interfere with the respiration of the patient, and there is danger of croupous laryngitis, I immediately order vapor inhalations and insist upon their continuance until the laryngeal symptoms shall have subsided. Sometimes this subsidence will take place within two or three hours, and, again, not until after two or three days. I desire to impress upon you the value of vapor inhalations in the treatment of the laryngeal and bronchial complications of measles. I have come to regard them as of great value.

When catarrhal pneumonia is developed it is to be treated in the same manner as catarrhal pneumonia developed under any other circumstances; the patient should be sustained by the free use of stimulants.

Pulmonary complications in measles are often the result of exposure to sudden changes in temperature; the severity of catarrhal symptoms will always be increased by such exposure, therefore it is of great importance in the management of a case of any type of measles that the patient should be protected against such exposure.

When there is great restlessness during the fever of invasion, or during the early period of the eruptive stage, small doses of opium, in the form of Dover's powder, may be administered with marked benefit.

The management of the different varieties of measles will be indicated by the general condition of the patient. In the ulcerative, hemorrhagic, and typhoid varieties, the free administration of stimulants should be early commenced. Usually in these varieties there is great prostration, and the thing to be accomplished is the support of your patient.

GERMAN MEASLES, or *Epidemic Roscola*.—Before leaving the subject of measles I will call your attention to an affection which has recently received the name of *German measles*. It is commonly known by the term *roscola*, or *mock measles*. It has been regarded by some as a modified form of measles; by others as a modified form of scarlet fever; again it has been thought to be a combination of the two diseases.

Some writers maintain that we are not justified in calling this type of measles an independent and specific disease, but that it may embrace any blotchy exanthemata, from the appearance of which we are unable to determine what we shall call the disease; whether scarlet fever, or measles, or urticaria, etc.

Later German writers regard it as an independent affection, a specific, acute, and contagious eruptive fever, and have given to it the name of *rubeola*.

I am disposed to regard it only as a different type of measles from that which ordinarily prevails, and by way of distinction will call it *German measles*, or *epidemic roscola*.

MORBID ANATOMY.—This affection must be regarded as one of the mildest of eruptive fevers. It has prevailed epidemically and endemically. The study of its morbid anatomy has been almost exclusively restricted to the eruption. This is an exanthemata consisting of irregular spots, or hyperæmic blotches, varying in size from a pin's head to a large pea, usually slightly elevated, so that when the hand passes over them the surface of the skin feels somewhat rough. Sometimes these spots occasion intense itching; they are quite distinctly separated by healthy skin, and disappear under pressure. As a rule, even at the acme of the development of the eruption, their color is a "pale rose red," paler than the intense red of the eruption of scarlet fever, or the peculiar bluish hue of the eruption in severe cases of measles. The eruption can readily be recognized. It is seen upon all parts of the body, but is most abundant upon the face and trunk. The spots are usually discrete; they often lie crowded closely together, but they are not confluent.

The eruption is exceedingly fugitive, rarely remaining visible more than twenty-four or forty-eight hours. It may continue visible for three or four days. The period of its most marked development may be only a few hours—twelve hours is the limit. In some cases there is slight desquamation; in most cases the eruption disappears, and leaves no trace, except in occasional instances, when there is a transient and yellowish discoloration of the skin. Some writers affirm that the eruption may disappear and reappear alternately for several days, and when it has finally disappeared the disease has terminated, and there is nothing to fear from complications or sequelæ. In certain rare cases vesicles resembling miliaria may be developed upon the hyperæmic spots, especially upon the back; doubtless these are chiefly due to external conditions.

ETIOLOGY.—Doubtless this disease is a contagious affection. Nothing is known concerning the nature of its contagion. It is essentially a disease of childhood. In persons more than forty years of age its development is of very rare occurrence. It is conveyed from one person to another in the same manner as measles. It has been thought by some that women were more susceptible than men to the influence of the contagion, and that high atmospheric temperature has a great influence in its development.

SYMPTOMS.—Epidemic roseola is so mild an affection, that it is questionable whether it has an invasive stage. The duration of the stage of incubation has not been determined. Generally, the symptoms which manifest themselves two or three days before the appearance of the eruption are much less marked than they are in any other eruptive fever. Perhaps in many cases they escape notice. Quite frequently the eruption is the first symptom of the disease. In some cases there may be nothing more than a feeling of discomfort. In other cases the disease may be ushered in by vomiting, diarrhœa, and convulsions. In many cases, immediately preceding the eruption, and accompanying its appearance, there is well-marked fever, headache, loss of appetite, and sometimes noticeable prostration. When the eruption is regular in its appearance

it affects first the face and scalp, then gradually extends downward over the trunk and extremities. Usually, the development and speed of the eruption is rapid, perhaps no more than two or three days being occupied in its passage over the entire body. Its duration upon any one part of the body before it begins to disappear is not more than from twelve to twenty-four hours. Within forty-eight hours it has almost entirely disappeared. In the majority of cases the temperature does not rise more than $100\frac{1}{2}^{\circ}$ F. to $101\frac{1}{2}^{\circ}$ F. above the normal standard. It may rise from 102° F. to 104° F. During the second day of the disease the temperature begins to fall. Sometimes it reaches the normal standard within twelve hours, occasionally not until the third day. Sometimes it reaches it by crisis, at other times by gradual descent.

The pulse increases and diminishes in frequency according to the rise and fall of temperature.

The tongue is usually covered with a whitish coating, is dotted here and there with red and swollen papillæ. The mucous membrane of the fauces is generally congested, and the tonsils moderately swollen; there may be some soreness of the throat.

The mucous membrane of the air-passages is usually in a condition of mild catarrh, consequently at the onset of the disease sneezing and coughing are frequently present, but they are less marked and are of shorter duration than in the ordinary type of true measles.

Suffusion of the eyes with congestion of the conjunctival vessels is rarely present; there may be a slight degree of photophobia. The face and eyelids are usually slightly swollen at the time the eruption makes its appearance, but this swelling rapidly disappears.

In most cases, there is moderate swelling of the lymphatic glands of the neck, and enlargement of the glands at the nape of the neck. Moderate enlargement of the occipital glands may continue for a number of days. Suppuration of lymphatic glands has not been observed. The urine is usually normal; it may, however, contain an abnormal amount of the chlorides.

You have already learned the fact, that when this disease runs its regular course, it is exceedingly mild in character. So mild, that children generally dislike to remain in bed, and prefer to be out-of-doors and at play.

DIFFERENTIAL DIAGNOSIS.—One of the prominent features of this disease is the close resemblance which its eruption bears to that of measles. In certain cases it may be impossible by the eruption alone to make a differential diagnosis. When the eruption of measles is not typically developed, a complete history of the case must be taken into consideration. When this has been done, usually there is no great difficulty in arriving at a correct diagnosis. Perhaps, that which will best aid you in making a differential diagnosis between roseola and measles is the fact that an attack of one does not protect against the other, any more than does an attack of varicella protect an individual from an attack of variola. This fact certainly establishes the non-identity of the two diseases.

It has been questioned whether a person may not have a second attack of epidemic roseola. The latest observations go to prove that a second attack of roseola is of as rare occurrence as a second attack of measles or scarlet fever. Again, the evidence seems most conclusive that an attack of this disease does not protect an individual against the contagion of scarlet fever; nor does an attack of scarlet fever protect one against the contagion of roseola. An individual may have an attack of German measles very soon after he has been ill with measles or scarlet fever.

PROGNOSIS.—The prognosis is always good. Complications rarely occur. When they do, they are usually pulmonary affections.

TREATMENT.—The treatment of this affection simply consists in protection against exposure. Tepid sponging will relieve troublesome itching, and reduce fever. Regulate the diet, and carefully watch the catarrh of the air-passages. In some cases, a mild course of tonic treatment may be beneficial. As a rule, convalescence is rapid, and is completed without hindrance.

MILIARY FEVER.

This form of fever cannot strictly be regarded as a contagious disease, but it so frequently prevails in connection with measles and scarlet fever, and has apparently so many elements of contagion, that I have included it in the list of contagious fevers.

Some deny its existence as a distinct fever. Writers have described it under the names of *sudamina*, *sudoral exanthema*, *miliaria alba*, etc. I shall adopt the name of miliary fever.

Several diseases which are accompanied by sweating, and which exhibit a tendency to the formation of miliary vesicles, have been called miliary fever. Until the occurrence of the severe epidemic of the disease known as the "English Sweating Sickness," its specific type was not recognized. It has prevailed epidemically over limited areas, in Belgium, France, England, Germany, Italy, and Austria.

In some of these epidemics one-fifth to one-tenth per cent. of the whole population of the invaded district has been attacked by the disease. The average duration of the epidemics has been from three to four weeks, occasionally they have lasted from three to four months.

MORBID ANATOMY.—Few post-mortem examinations have been made, and those few have failed to reveal any characteristic lesion.

During life the blood is thin, of a bright-red color, and coagulates imperfectly; after death it is thin and dark-colored.

Generally, the internal organs present evidences of hyperæmia. The mucous membrane of the air-passages is red and frequently covered with reddish mucus. The lungs and liver are generally filled with blood; the latter is softer than normal. The spleen is always enlarged and soft. Some observers have reported the kidneys to be in a normal condition; other observers have reported them to be in a condition of congestion. The mucous membrane of the stomach and intestines is usually reddened, and presents here and there red spots. Occasionally these spots are very

numerous, and vesicles are sometimes seen in the small intestines. By some these vesicles have been supposed to be swollen, solitary follicles; by others they have been thought to be distinct miliary vesicles, similar to those which are seen upon the surface of the body. Superficial ulcers are sometimes seen, especially in the region of the ilio-cæcal valve.

The miliary vesicles which are seen upon the surface of the body, and the cutaneous eruption, are developed because the secretion of the sudoriferous glands cannot escape.

The escape of the contents of these glands may not take place for two reasons: First, the gland-ducts may become obstructed. Second, the secretion may be so abundant that it cannot be transmitted by the gland-duct.

In either case, the secretion emerges under the epidermis around the sweat-duct, and, as the scales are lifted up, a small clear vesicle is formed. The liquid contained in the vesicle at first is transparent, has an acid reaction, and is said to contain free nuclei-cells, which have three or more nuclei; these nuclei remain visible after the cell membrane has been destroyed by the addition of acetic acid.

It has been claimed that the virus of the disease is contained in these polynucleated cells. After death, the skin becomes œdematous, and very soon the odor of decomposition is perceivable.

ETIOLOGY.—It was formerly supposed that miliary fever was indirectly induced by scarlatina, the puerperal condition, variola, vaccinia, typhus fever, and other diseases, and that it was not a distinct disease arising from some constitutional cause. The prevalence of this fever in connection with these diseases gave rise to this supposition.

Epidemics of this disease have generally prevailed during the spring and summer months; from this fact one would be led to think that there is some atmospheric condition peculiar to these months. Again, the disease has most frequently appeared in warm, moist weather, and from this fact it has been supposed that some peculiar condition of the soil is necessary to its development. Certain epidemics

have shown a close connection with contaminations of the soil, such as arise from neglect of drainage, collections of refuse, etc. Doubtless, such conditions of the soil may increase its severity, and cause it to prevail more extensively, but facts do not prove that, directly or indirectly, they cause its development.

The disease usually attacks healthy adults, and occurs more frequently among females than males. It attacks all classes, and its spread does not seem to be affected by crowding.

It can hardly be regarded as a contagious disease, in the sense that it can be communicated directly from the sick to the well. It does not seem to be well established that the disease can be developed by inoculation with the contents of the vesicle, notwithstanding it has been supposed that certain cells in the fluid hold the contagion of the disease.

The infrequency of the simultaneous occurrence of miliary fever, with epidemics of measles or scarlet fever, is unfavorable to the theory that there is a specific relationship between the poisons of these diseases.

The view that there is an intimate relationship between cholera and miliary fever has been accepted by some writers, and the accession of the latter during the course of the former has been supposed to exert a favorable influence over the course of the disease; the opposite, however, does not appear to hold good, but, on the contrary, favors a fatal termination. Much remains to be learned in regard to the relationship existing between miliary fever and the other diseases which we have mentioned.

The etiology seems to be mainly speculative and theoretical.

SYMPTOMS.—The average duration of the disease is from five to eight days.

It has three stages: First, *the stage of invasion*; second, *the stage of sweating*; third, *the stage of eruption and desquamation*.

The stage of invasion.—The average duration of this stage is from forty-eight to seventy-two hours. It is characterized by an excessive irritation of the skin, thirst, gen-

eral lassitude and headache. There is also more or less febrile movement.

Some writers mention a feeling of suffocation, which is usually preceded by a sense of oppression at the epigastrium. These are the characteristic symptoms of the stage of invasion.

The stage of sweating.—This stage is usually ushered in by rigors ; rarely, by a well-marked chill. The characteristic symptom of this stage is profuse and persistent sweating. The sweating is accompanied by a prickling sensation of the skin, distress, and a sense of compression at the epigastrium, by more or less violent palpitation of the heart, with precordial pain. Usually the sweat appears on all parts of the body at the same time. Sometimes it appears first upon the head and breast, then gradually descends, and soon becomes so abundant that every article of clothing, bed-clothes, and bedding, becomes saturated.

The pulse sometimes reaches 140 beats per minute, the temperature rises to 103° F., 104° F., or 105° F., and the skin, notwithstanding the profuse perspiration, feels extremely hot.

During this stage the headache and the sense of suffocation increase, the epigastric and precordial pain, and the palpitation increase in severity, and sometimes become alarming, although the most careful physical examination fails to discover any lesion in the heart or lungs to account for them. The respiration becomes rapid, often irregular and intermittent. Irregular exacerbations, or even intermissions, in these symptoms may occur, but, as a rule, they continue without abatement until the vesicle appears on the third or fourth day of the disease.

THE STAGE OF ERUPTION.—This stage is characterized by the appearance of a rash. It is first seen upon the neck and breast, then upon the back and extremities, sometimes upon the mucous membrane of the mouth, nose, and conjunctiva, sometimes upon the abdomen and scalp. This eruption consists of irregularly shaped spots, three or four mm. in diameter. In some cases they stud the skin so thickly that it appears like an uniform sheet of vivid redness.

After the lapse of a few hours, vesicles can be seen in the centre of these spots; perhaps, at first, they are so small as to necessitate the aid of a lens to discover them. These vesicles rapidly increase in size, and may reach the size of a millet-seed or a small pea. The contents of these vesicles have already been described.

Occasionally, as the eruption appears, all the constitutional symptoms are increased in severity, but, usually, they are modified and disappear either suddenly or gradually after its development. In the milder cases the vesicles only, without the efflorescence, are seen.

Vomiting is rarely present, although nausea is a common symptom, as is also constipation. The urine is usually scanty and high colored; in some cases there is suppression of urine. Occasionally, during the stage of eruption, profuse secretion of urine takes place. This has been regarded as a favorable symptom.

The vesicles, clear at first, soon become opaque and yellowish, continue for two or three days, then burst and begin to fall off in scales. Desquamation is usually completed within forty-eight hours, but convalescence is often quite protracted on account of the debility and emaciation. Such is a brief description of miliary fever, when it runs a regular course, but there are certain variations in the development of the symptoms which should be noticed. In the severest form of the disease, the temperature may rise to 107° or 108° F., and there may be a sense of suffocation and raging delirium. Again, there may be absence of the eruption, sweating, and convulsions followed by death. Occasionally sudden and fatal collapse follows the sweating stage.

The typhoid condition may be developed in the sweating stage, and may be attended by black sordes upon the teeth and tongue, epistaxis and uterine hemorrhage, and may terminate in death, without any considerable anatomical changes recognizable after death.

Complications are not of frequent occurrence. Occasionally there is bronchitis, pneumonia, and angina.

Relapses are of common occurrence, but recovery generally takes place after a short relapse.

DIFFERENTIAL DIAGNOSIS.—Miliary fever may be confounded with measles, with typhoid fever, and with dengue fever. The profuse sweating, the prickling of the skin, the intense oppression at the epigastrium, the sense of suffocation, with precordial pain, and the peculiarity of the eruption, are sufficient to distinguish it from measles, from intermittent fever (although a decidedly intermittent type of the disease sometimes prevails), and from typhoid fever.

When the disease prevails epidemically, the diagnosis cannot be difficult.

PROGNOSIS.—When the disease runs a regular course, with only a moderate degree of severity, the prognosis is good; whereas, great severity of the febrile symptoms, exceptionally profuse sweating, and increasing sense of constriction of the chest, with suffocation, render the prognosis unfavorable. The accession of profuse hemorrhages, coma, convulsions, active delirium, or symptoms of collapse, render the prognosis unfavorable.

The severity of the symptoms is usually mitigated when the eruption makes its appearance, and death rarely occurs after that stage is reached. If fatal termination is reached, it usually takes place during an exacerbation, prior to the appearance of the eruption.

In some epidemics, the mortality has been very great; in other epidemics the disease has been mild in character.

The character of the epidemic affects the prognosis.

TREATMENT.—At one time diaphoretics were employed in the treatment of this disease, on the supposition that the sweating and eruption were critical manifestations, and must be aided by all possible means.

The sense of suffocation, with that of constriction of the chest, was thought to indicate blood-letting; but it was soon decided that loss of blood aggravated rather than improved the patient's condition.

Antispasmodics, nervines, quinine, emetics, and counter-irritants, at different times have formed the basis of various plans of treatment. Of late, subcutaneous injections of morphine have been used with advantage. Sinapisms and blisters have been employed for the relief of the sense of

constriction in the chest, and for the epigastric and precordial distress, with benefit to the patient.

It is now acknowledged that the administration of purgatives in large doses should be carefully avoided, as well as bloodletting, general or local.

At present the expectant plan of treatment is regarded with most favor. It chiefly consists in the use of cooling drinks, aromatic teas, acidulated water, sponging with warm water, or the employment of warm baths. It has been thought that the addition of alum or vinegar to the water used for sponging or bathing is beneficial.

In the treatment of this affection, quinine seems to be regarded with almost universal favor. If restlessness is persistent, opium, ether, and antispasmodics may be employed in moderate doses, carefully watching the effect produced. The patient should be surrounded by proper hygienic influences, his diet should be moderately nutritive, and, in those cases in which convalescence is tedious, a steady and continued tonic treatment is indicated.

In the severest form of the disease stimulants may be employed with benefit.

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